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VETERINARY MEDICINE

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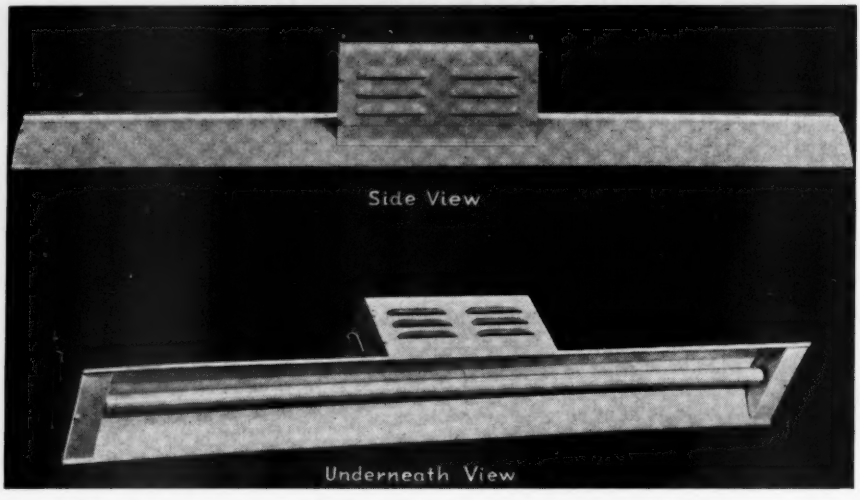

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The consumption of cheese has doubled in this country since 1926; the average per capita consumption being now six pounds annually. However, the annual per capita consumption in European countries ranges from 12 to 14 pounds; a figure that if attained in the United States will mean a vast new market for the dairy industry.

Eleven million cattle and sheep are grazed on the 134,000,000 acres of federal ranges of 11 western states. One of the problems of range management is the elimination of wild horses. A decade ago these animals were estimated at 150,000. There are believed to be not more than 25,000 at present on ranges adapted to cattle grazing. Their complete extinction is expected in the near future.

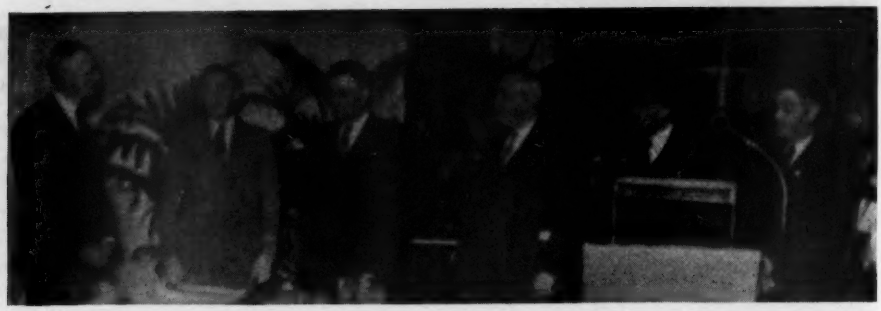
The cost of distributing foods has increased more than 30% in the last score of years. According to the U. S. Department of Agriculture, the percentage of the consumer's dollar paid for transportation, processing, and all other marketing costs on 58 representative foods rose from an average of 45% in the eight-year period 1913-20 to 54% in the decade 1921-30 and to 59% in the 1931-38 period. Marketing efficiency for agricultural products has received increasing study and research during this period but such studies have been barren.

Shorthorn cattle were first imported into the United States 125 years ago.

At the U. S. Range Live Stock Experiment Station, Miles City, Mont., the level of fertility of the stud was raised from 50% to 80% by making an accurate check on the ovarian cycles in the 70 mares comprised in the stud and timing insemination to coincide closely with ovulation.

In view of the large increase in the number of hogs it is expected will be slaughtered and the probable low price of lard, live stock papers are urging swine growers to contribute five cents from the price of each hog sold to a fund for an educational campaign to increase the use of lard. It is estimated that such a campaign would add a dollar to the value of each hog.

Fontana Farms, near Fontana, Calif., said to be the largest hog farm in the world, comprises 540 acres with 320 acres actual hog area. An average of 55,000 hogs are maintained on the ranch, which employs 130 men. The hogs are fed garbage from Los Angeles, brought to the ranch by rail and unloaded with a steam shovel directly onto the feeding floors. The animals are finished on grain and 4,000 to 5,000 marketed annually at Los Angeles packing houses.



INSTALLATION OF OFFICERS AT THE 43RD ANNUAL U. S. L. S. S. A. MEETING
Left to right: J. L. Axby, State Veterinarian, Indianapolis, Ind., retiring president, who conducted the installation ceremony; Mark Welsh, State Veterinarian, College Park, Md., secretary-treasurer; H. D. Port, State Veterinarian, Cheyenne, Wyo., president; E. A. Crossman, Needham, Mass., 1st vice-president; I. McAdory, State Veterinarian and Dean of the Alabama Polytechnic Institute, Auburn, Ala., 2nd vice-president; W. A. Hendricks, State Veterinarian, Salt Lake City, Utah, 3rd vice-president.

Horse of the Year

Country Life has announced as its Horse of the Year Challedon—"a Thoroughbred of courage, endurance, stamina, the ability to race hard under all conditions, the willingness to go on . . . the leading money-winner of the world for 1939." Challedon was chosen in preference to the brilliant two-year-old Bimelech because the latter has yet to show development as well as precocity. Challedon's record to date shows 21 starts; 13 firsts, two seconds, four thirds, twice unplaced. His sire was Challenger; his dam, Laura Gal.

* * * *

Hereford cattle were first imported into the United States in 1817. In 1825, Henry Clay took the first of this breed to Kentucky.

At the Smithfield live stock show in London, Aberdeen-Angus or Aberdeen-Angus crosses have won 92% of the prizes for the last 25 years. At the "International" in Chicago,they have won 75% of the prizes.

* * * *

The phenomenally rapid acceptance of soy bean oil meal by cattle and sheep feeders during the last few years is based on the following sound facts:

1. Soy bean oil meal is the most highly digestible vegetable protein concentrate we have today.

2. As a general rule the price per unit of digestible protein is lower than in other feeds of this class.

3. Soy bean oil meal is not unduly laxative when fed in the amount necessary to balance farm-raised grains.

4. Soy bean oil meal is more palatable and produces faster gains than other protein concentrates or soy beans.

5. Cattle fed soy bean oil meal in most of the experiments had a better finish and brought a better price than those fed cottonseed meal or soy beans.—*Lyman Peck.*

* * * *

Common goats produce less than 500 pounds of milk containing 25 pounds of fat during an average lactation period of 240 days. During a comparable period improved goats will produce 1,500 pounds of milk. Goats have produced as high as 4,000 pounds of milk in the year. The feed requirement to produce a pound of goat's milk is comparable to that required for an equal quantity of cow's milk.—*J. C. Marquardt,* in the *Goat World,* 24:11.

* * * *

Crystal Helen, 4½-year-old Swiss Toggenburg, has produced over 10,000 pounds of milk in her lifetime—nearly 75 times her own weight. She holds the world's record for milk production for a pure Swiss Toggenburg, with 3,371.4 pounds in 12 months in her last lactation. She was started on another official test January 15, 1939, and up to October 1st had produced 3,316.6 pounds. Crystal Helen has also produced seven kids; two at her first freshening, two at her second and three at her third. All are alive and well except one.

Live Stock Becoming Better and Healthier

In a report to the Secretary of Agriculture on progress in live stock research and the control and eradication of live stock diseases for the fiscal year ended June 30, 1939, John R. Mohler, Chief of the Bureau of Animal Industry of the U. S. Department of Agriculture, points out that tuberculosis among cattle has been practically eradicated from 47 states, with the work nearing the same goal in California, the forty-eighth state; and that tick fever of cattle is now confined to limited areas in southern Florida and southwestern Texas, aggregating less than 1% of the area formerly infested with cattle ticks. Bureau and State officials are now combating Bang's disease on a nation-wide scale. Of the 7,600,000 cattle to which diagnostic agglutination tests were applied during the year, approximately 2.9% were slaughtered as reactors.

In the field of research, Doctor Mohler tells of the discovery that the non-toxic and comparatively inexpensive chemical, phenothiazine,[1] is highly effective for removing certain internal parasites of sheep and swine. The Bureau has applied for a public-service patent, to make the product available at minimum cost.

In the field of animal husbandry research, the Bureau's investigators are developing the Southdale from crosses of the Southdown and Corriedale breeds. The Southdown is noted for the quality of its meat but produces a light, short fleece, whereas the Corriedale produces mutton of low quality but a superior fleece. The Southdale is being developed to combine the desirable qualities of both breeds.

Recent studies by Bureau animal husbandmen have developed a record-of-performance test for beef cattle to be used in "proving" herd sires. It consists of two simple body measurements—the height at the withers and the heart girth—plus a record of feed consumed in reaching a certain weight, the former giving an index to carcass quality, and the latter telling how efficiently the animal puts on weight.

The Bureau's investigators have made progress in the breeding of a small-type turkey to meet the demand for a bird suitable for small families. Young hens of the new type weigh, when killed and plucked, from 6½ to 9 pounds and young toms from 10½ to 15 pounds. The birds have a compact body, large proportion of breast meat, and relatively short legs.

The Bureau continued its supervision of the National Poultry Improvement Plan as a means of improving the breeding and production qualities of poultry and controlling mortality from pullorum disease. This plan, conducted in coöperation with 44 state agencies, has been in operation since July, 1935. Participants in 1939 included 2,035 commercial hatcherymen whose plants had a total capacity of nearly 76,000,000 hatching eggs. In all, there were more than 8,000,000 breeding birds in 42,461 flocks that supplied eggs to these hatcheries.

Each year approximately 65,000,000 cattle, sheep, and hogs, sent to the principal markets, are examined by trained veterinary inspectors. This supervision makes possible the detection of contagious live stock diseases in animals intended for reshipment. Railroad cars and other vehicles in which diseased live stock are shipped are cleaned and disinfected as an additional safeguard.

As in former years, the Bureau conducted the Federal inspection of meat and meat food products on an extensive scale. This inspection is conducted at all establishments that ship their products in interstate or foreign commerce. During the fiscal year more than 71,000,000 food animals were slaughtered under Federal inspection at 684 establishments located in 249 cities and towns. Inspection also extended to the curing, canning, and other processing of more than 8,400,000,000 pounds of meat and meat food products.

[1] *Vet. Med.* 34:7, pp. 440-443; 35:1, pp. 24-29.

The Mineral Balance in the Blood of Ruminants*

By H. H. GREEN, Weybridge, England

Introduction

At the outset a mild protest is necessary against the common use of terms such as "calcium deficiency," "magnesium deficiency," and "phosphorus deficiency" to express physiological states more accurately described as "hypocalcemia," "hypomagnesemia" and "hypophosphatemia." Loose usage in this connection is likely to cause confusion between low mineral content in the food and low mineral content of the blood, and suggest, for instance, to the lay mind at least, that low blood minerals can be corrected by increasing the mineral content of the ration—a palpable fallacy; or that mineral deficiency can always be diagnosed by blood analysis—another fallacy.

Although the low level of a particular mineral in the blood *may* be related to a corresponding deficiency of that mineral in the food, or in the animal body as a whole, there is no necessary relationship at all, even in advanced cases of certain well-known mineral deficiency diseases.

Calcium and Phosphorus

For example, in osteofibrosis of the horse, a disease easily induced in six to 12 months, according to age, by feeding exclusively on bran, a ration very low in calcium and very high in phosphorus, skeletal changes may advance to the point at which fractures occur, without noteworthy alteration in the blood level of either calcium or phosphorus. The disease cannot be diagnosed by blood analysis, although the deficiency of calcium in the food is known and the drain upon the skeleton is obvious, radiologically or clinically. X-ray pictures show the disappearance of calcium from the bones, but there is no material hypocalcemia. A small supplement of chalk, supplying calcium only, is sufficient to pack the minerals back into the skeleton with great rapidity, but again without material alteration of the blood calcium.

On the other hand, if the ration be made adequate in calcium but deficient in phosphorus, the blood picture of any animal reacts rapidly. Lowered inorganic phosphate is detectable even after a few weeks and the figure falls to extraordinarily low levels long before definite clinical symptoms are manifested. In aphosphorosis of the bovine (the "styfsiekte" of South Africa), a true osteomalacia from a histological point of view, the inorganic phosphate may fall to one-fifth the normal value and may even reach this low level when the deficiency of phosphorus is only sufficient to cause retarded growth and reduced fertility, without specific signs of osteomalacia at all.

What happens to the mineral balance of the blood if an animal is fed a ration low in both these elements? Much depends upon the species of animal and its age, particularly upon the stage of development of the skeleton. With lambs, kids and calves there is the usual rapid fall of inorganic phosphate in the blood but only a slow, uncertain fall in calcium which may or may not be accompanied by clinical manifestations of hypocalcemia, and for which the explanation is not necessarily the mere shortage of calcium but also involves consideration of endocrine factors. With adult cattle, however, the fall in calcium may be negligible. If a two-year-old heifer is fed on such a ration, plasma phosphate falls rapidly as usual, but plasma calcium tends to be arrested at a definite lower physiological limit. After a year or so, when the animal is well on the way to osteomalacia, the inorganic phosphorus may have fallen from 5mg% per 100cc to 1mg%, whereas the plasma calcium may have fallen only from 10mg% to 8mg%—a fall of 80% in one

*Presented at the 57th Annual Congress of the National Veterinary Medical Association of Great Britain and Ireland, July 23-28, 1939.

case but only 20% in the other. This "physiologically low" but not "pathologically diagnostic" level of blood calcium may be retained throughout another year and still be shown when more than half the skeleton is gone and death from extreme osteomalacia ensues.

Contrast this with the behaviour of a cow abundantly fed on both calcium and phosphorus, with practically normal blood levels of both elements shortly before calving, yet developing milk fever within a few hours, with dramatic fall of plasma calcium from 10mg% to 3mg% and lesser fall of plasma phosphate. Remedy this condition by the nervous stimulation of udder inflation without altering the ration, and the cow proceeds to maintain a normal level of blood calcium despite the enormously increased demands for that element imposed by high secretion in the milk.

The reason for this varying behavior is, of course, now well known. The level of plasma calcium is under the influence of a rigorous physiological controlling mechanism, whereas the level of plasma phosphate is not. It is true that on normal dietaries the level of inorganic phosphate of the blood tends to remain at a fairly constant level but there is no rigorous endocrine control, arbitrary fluctuations are considerable, and fall is almost unrestricted under conditions of dietary deficiency. While the skeleton is rapidly parting with both calcium and phosphorus on a diet deficient in both, the blood level of the former is maintained fairly near the normal level, while that of the latter is not. If the skeleton is breaking down because of calcium deficiency associated with high dietary phosphorus, there is, of course, no fall of plasma phosphate and, as in the case of equine osteofibrosis, the level of both elements does not deviate materially from normal.

The generally accepted physiological mechanisms for control of plasma calcium are the parathyroid glands operating in conjunction with the sympathetic nervous system through ganglia in the region of the carotid sinus and glomus (Hering and Heymans), and drawing on skeletal reserves of calcium when absorption of calcium from the alimentary tract is insufficient; but other endocrine glands, notably the anterior lobe of the pituitary, also play an important part.

Magnesium

For the element magnesium there is also some definite nervous endocrine control, although the mechanism is less clearly understood. The main evidence (Cannavò and others) suggests the anterior lobe of the pituitary, although interaction with other glands, including the pineal and the parathyroids, probably occurs. Injection of anterior pituitary extract has been recorded as increasing the blood magnesium in dogs, rabbits, cats and man, although this has not yet been confirmed for the bovine subject. Conversely, fall of blood magnesium followed by death has been recorded after x-ray destruction of the pituitary (Cannavò and Beninato). Pineal extract is said (Fiandaca) to abolish the magnesium-elevating effect of subsequently administered anterior pituitary extract. Subnormal values for blood magnesium have been reported in human parathyroidism but experimental parathyroidectomy has not produced hypomagnesemia.

Nutritional requirements of magnesium are very low and if we discount the explanation of Huffman for "milk tetany" in calves, no naturally occurring magnesium deficiency disease has been reported in animals. Magnesium deficiency has, however, been produced in rats and dogs on specially prepared synthetic diets exceedingly low in magnesium, the main symptoms being vasodilatation, cardiac arhythmia, hyperirritability and convulsions. Serum magnesium values fall but tend to rise temporarily after a convulsive attack, presumably at the expense of skeletal reserves. Death usually supervenes after one or two convulsive attacks. If deficient rats are allowed to recover by supplementing with magnesium and are then put back on to the deficient diet, the tendency is to develop a state of chronic malnutrition without convulsive attacks—presumably associated with chronic hypomagnesemia.

According to Greenberg, lesions in the mesencephalon or the pons are detectable in magnesium-deficient animals. According to Moore, calves on a magnesium-low diet showed deposition of calcium salts in the yellow elastic fibers of the endocardium, aorta and larger arteries, but it seems doubtful whether the sole cause was dietary deficiency of magnesium.

The main point of interest, however, from a veterinary point of view is that just as hypocalcemia can appear in the bovine species (and in others, including the ovine and the equine) on diets high in calcium, so hypomagnesemia can appear on diets adequate in magnesium. Indeed, the magnesium content of grass and most cattle foods is so high in relation to real minimum requirements that there can be no question of dietary deficiency in the ordinary sense of the term in hypomagnesemic tetany (lactation tetany or grass tetany) of farm animals. The stemming of seasonal fall of plasma magnesium by feeding additional easily available magnesium compounds should be interpreted, when it appears to occur, as a sort of "flushing effect," rather than as meeting an existing deficiency.

The primary disturbance in hypomagnesemic tetany is regarded as dysfunction of the physiological mechanisms controlling the level of blood magnesium, although the exciting cause is not usually parturition as in the case of the physiologically parallel hypocalcemic condition commonly manifested as milk fever. Indeed, the stimulus of parturition usually has the effect of raising rather than lowering blood magnesium, although notable exceptions occur, particularly if the animal has a previous history of lactation tetany (Weybridge data).

In the hypocalcemic condition the fall of blood calcium is sudden and the rise on recovery rapid. The fall of blood magnesium in hypomagnesemic tetany may also be sudden in both cattle and sheep, with fairly rapid return to normal after recovery from convulsive attack but in the commoner bovine manifestation (grass tetany) a slow fall occurs over several days or weeks before a presumably sharper second fall preceding a convulsive attack, and the return to normal after recovery may be equally slow. Unlike the simple hypocalcemic condition, uncomplicated hypomagnesemia is not usually accompanied by hypophosphatemia unless the plasma calcium is also well below normal.

It may be added that cases of bovine hypocalcemia remote from calving date also occur, in which clinical symptoms of atypical milk fever are preceded by slow fall of blood calcium over many weeks, followed by sharper fall and, on recovery, equally slow return to normal. Some cases of depressed calcium level show no specific clinical symptoms and hypocalcemia would pass undetected except by blood analysis. One cow under recent observation, at a time remote from calving, showed a serum calcium of 6mg%, with normal magnesium, for several weeks until disposed of as an uneconomic animal on grounds of ailing and progressive unthriftiness.

The case of comparatively slow fall of blood magnesium over days or weeks, with latent tetany easily precipitated in convulsive form by a variety of exciting causes, is the type most familiar to practitioners, but a type of seasonal hypomagnesemia is

also common in which the fall of blood magnesium occurs very slowly over several months and the animal seems to gain time to readjust its neuro-muscular responses in such a way that no clinical symptoms, nor alteration in physical condition or general health, are shown at all. The upward return to normal summer values is equally gradual and the fact that the animal has passed through a hypomagnesemic phase, with blood values as low as those commonly encountered in acute convulsive tetany, would never be detected except by random blood analyses.

Artificial Change of Blood Calcium and Magnesium. — Before considering newer views on "milk fever" and "grass tetany" in more detail, something may be said on the effect of experimentally varying the levels of calcium and magnesium in the blood stream.

Common normal values are about 10mg% calcium and about 2.3mg% magnesium, i.e., a ratio of about four or five to one. In milk fever the calcium value is commonly halved and the magnesium somewhat raised, giving a ratio of about two to one. In grass tetany the magnesium value is commonly about one-third normal and the calcium somewhat reduced, giving a ratio of about 10 to one or 14 to one.

If in the normal bovine animal the calcium figure be doubled or trebled by intravenous injection of a calcium salt the magnesium value remains unchanged and no particular symptoms are shown, although the ratio now simulates that of grass tetany. Apparently absolute values, quite as much as "ratio" or "balance," control neuromuscular responses. The artificially high calcium value falls very rapidly by distribution from the blood stream into the tissues, approaches normal in an hour or so and is back to the original level in six to eight hours. The period of markedly raised calcium is very brief — somewhat longer if the calcium be given as borogluconate in more massive subcutaneous doses.

On injection of a magnesium salt the same sort of thing occurs, except that symptoms of narcosis may be shown if the dose is high. There is no change of blood calcium and the artificially raised magnesium level rapidly returns to normal by distribution over the tissues. The period of elevated magnesium is very brief—again lengthened if more massive doses are given by the subcutaneous route. No symptoms are noticeable if the normal value is momentarily doubled or trebled by intravenous injection, but above that reduced response to stimuli may be observed and at a level of about 16mg%, or about seven times the normal value, complete temporary anesthesia supervenes. The anesthesia may be prolonged by successive subcutaneous injections of massive doses since the blood value is thereby maintained at a high level, but there is danger of exitus.

If sodium oxalate be cautiously injected intravenously so as to reduce ionized calcium of the blood stream, leaving magnesium unchanged, the general effect is to reduce sensitivity to mechanical stimuli, induce paresis of the extremities, muscle twitchings and increased respiration. If an injection of a magnesium salt follows the oxalate at a short interval the effect is to heighten the narcosis. In certain experiments of Sjollema on calves only a few days old, narcosis, with moderate intravenous injections of magnesium chloride or sulphate was only induced after preliminary injection of oxalate to reduce the level of plasma calcium.

There is no known method of experimentally reducing the level of blood magnesium in cattle, other than possible trial with massive doses of the gonadotropic hormone of the anterior pituitary along the lines of the experiments of Cannavò on laboratory animals, but if it could be suddenly effected increased nervous hyperirritability would presumably ensue. The effect, however, would presumably depend upon the real level of reduced blood magnesium rather than on its ratio to normal calcium, since alteration of the ratio by elevating the calcium does not induce tetany. A normal blood magnesium with artificially reduced calcium would appear to simulate certain of the features of milk fever, although an artificially raised calcium with

normal magnesium does not simulate the features of grass tetany.

It is interesting to note in passing that along with the reduced calcium in milk fever the magnesium may be raised so definitely that Kloubeck even advanced a theory of "magnesium narcosis" to explain the comatose condition.

Milk Fever

The well-known hypothesis of "temporary hypofunction of the parathyroids" advanced by Dryerre and Greig in 1925 and supported by the contemporaneous demonstration of hypocalcemia by Little and Wright, seems to have held the field in England almost to this day, although considerably modified in Holland by the views of Sjollema and Seekles from about 1927 onwards, in which dysfunction of more than one endocrine organ, and associated disturbance of equilibrium of the sympathetic nervous system were predicated. The outlook reached by Sjollema by 1933 in regard to both hypocalcemic and hypomagnesemic conditions, is given in his comprehensive article, "Stoffwechselstörungen des Riñdes" (*Acta. Vet. Neerlandica,* Vol. 1, No. 2), a publication which has not received the attention it deserves in other countries. The Dutch article of Seekles in 1937 (*Tijdschr. v. Diergeneeskunde,* 64:866) substituted temporary hyperfunction of the anterior pituitary for hypofunction of the parathyroids and explained the complete syndrome of milk fever, including the frequently associated hypoglycemia and mild acetonemia, as due to a lack of balance between the various hormones secreted around the time of parturition.

Although milk fever is not specifically discussed by H. D. Kay in his review article on "Mammary Activity" (*Science Progress,* January, 1939), his general discussion of hormonal control lends color to the views of Sjollema and Seekles, and present knowledge of the inter-relationships of the ductless glands makes it probable that almost the whole "nervous-endocrine system" is involved.

Crude extracts of the anterior pituitary gland possess many hormonal activities, some of which may be separated by suitable fractionation methods. There is, for example, the fraction called "prolactin" which not only stimulates lactation but which causes a marked increase in weight of the crop glands of the pigeon. This can be further fractioned into a portion which stimulates the crop gland but is not associated with true lactogenic activity, and a fraction which has no crop-gland power but is strongly lactogenic. There is a "thyrotropic" fraction which stimulates the growth of the thyroid in immature animals, a "glycotropic" fraction which prevents the hypoglycemic effect of insulin, a "diabetogenic" fraction which induces the symptoms of diabetes in normal dogs, and several other fractions. The estrone secreted by the ovary controls the growth rate of the mammary gland but is inactive in this connection after extirpation of the pituitary and there is evidence, from implantation of pituitaries with and without previous injections of estrone, that the ovarian hormone acts indirectly by inducing the production of a "mammary-gland-growth-stimulating" hormone by the pituitary. Normal milk secretion depends not only on the presence of an intact pituitary but also on the adequate functioning of the thyroid and parathyroid glands. The thyrotropic hormone, or thyroxin itself, increases milk secretion of cows in declining lactation but not in cows at the height of lactation, when the production of thyrotropic hormone is at a maximum and the animal is on the verge of a hyperthyroid state. Estrone, on the other hand, inhibits lactation.

In regard to the initiation of lactation at least two factors are concerned, one which removes the inhibitory effect of the estrogenic hormones and the other an excitory internal secretion of the anterior lobe of the pituitary. Certain work suggests that the mechanical distension of the uterus may be a contributory factor in the prevention of lactation before parturition, and that although lactation is not dependent upon suckling, further liberation of lactogenic hormone is induced as a nervous re-

flex by the mechanical stimulation of the teats.

As Kay puts it, "the dairy cow is the creature of her pituitary and cannot rise above the functional efficiency of this gland."

When it is added that, according to Eggs, injection of the thyrotropic hormone may cause a fall in serum calcium, from 11mg% to 5.3mg% in the case of one of his experimental guinea-pigs, that injection of the gonadotropic hormone, according to Cannavò, causes a rise in blood magnesium, and that the glycotropic and ketogenic hormones influence the carbohydrate-fat metabolism, the familiar biochemical picture of milk fever begins to emerge from the anterior pituitary—the hypocalcemia, usually associated with definite hypermagnesemia, the frequent hyperglycemia, and the transient acetonemia which occurs in such a large proportion of milk fever cases at a level usually escaping clinical notice. The clinical picture arises, according to Sjollema, from a failure of the sympathetic and parasympathetic nervous systems to respond adequately to stimuli and work in harmony under reduced concentration of calcium ions.

Since certain aspects of pituitary function are known to fluctuate around estrus, increase during pregnancy, rise to a maximum during lactation and sink during nonpregnant dry periods, the occurrence of "atypical milk fever" at periods remote from actual parturition may find a similar explanation in temporary dysfunctions of an idiosyncratic gland from a variety of unknown causes.

Milk fever in the semi-wild cow bred under ranching conditions from stock of low milk yield is a rare phenomenon, but with the modern dairy cow, with its genetic strain for high lactation and associated hyperactive anterior pituitary, the act of apparently normal parturition often induces a biochemical syndrome carrying the animal to the verge of that shown in clinical milk fever—so that for every clinical case there are several more which just escape the pathological exaggeration of a physiological happening. Hypophosphatemia may be very marked around the hour of delivery, the blood calcium tends to fall slightly in late pregnancy and after parturition may sink to 7mg% without clinical symptoms, while the blood magnesium fluc-

Typical milk fever in a grade cow

tuates, usually in an upward direction. In the course of one to four days this mineral imbalance settles down to its normal equilibrium, sometimes with an intervening compensatory swing in the opposite direction, *e.g.*, the calcium may swing from 10mg% to 8mg% and overswing to 11mg% before steadying again at 10mg%.

If the post-parturient fall of blood calcium is still sharper, as a result of imbalanced secretion of the various pituitary hormones, ganglia of the nervous system become involved and the clinical picture of milk fever supervenes. If the parathyroids fail to respond by reversing the fall with sufficient rapidity the initial slight hyperirritability passes over into coma and the animal is caught in a "vicious cycle" from which it may not recover. If calcium is injected, in amount trifling in comparison with the enormous easily available skeletal reserves (at the disposal of the parathyroids), in such a way as to effect a temporary artificial rise in blood calcium, the ganglia in the carotid sinus and great arteries (alleged to be sensitive to low calcium) are stimulated, sympathetic tone is restored, the parathyroids given another chance to reassert control, and the anterior pituitary to behave itself with regard to the proper admixture of the various hormones it secretes. The artificial elevation of blood calcium is very brief (an hour or two in the normal animal) but rapid reassertion of natural physiological control, often in a

matter of minutes, prevents fall to the former hypocalcemic level, although the true normal value may not be reached for some days—sometimes with a compensatory overswing, as noticeable in some of the earlier post-recovery values found by Dryerre and Grieg.

A similar effect may be induced by injection of parathyroid extract; temporary elevation of blood calcium, presumably by abstraction from the skeletal reservoir, is followed by restoration of sympathetic tone and reassertion of authority by the normal physiological mechanisms for control of blood calcium. Again, time is the essence of the contract.

The effect of udder inflation is presumably due to nervous reflex action induced by pressure; that it is not due to economy in calcium from damming back the milk flow is indicated by the facts that the alveoli may be full of milk already, that the action is often very rapid, and that injection of air into the subcutaneous tissues of the neck is occasionally sufficient to achieve the same purpose. Indeed R. F. Montgomerie reported recovery in a stallion suffering from transit tetany, as following inflation of subcutaneous tissues. The observation that cows susceptible to milk fever, but milked right up to parturition, are more likely rather than less likely to escape recurrence of attack, is against any idea that calcium drain, as such, is an important factor; as is also the fact that cows suffering from osteoporosis as a result of low calcium rations are no more likely to contract milk fever than those receiving superabundance of mineral supplement. One of the cows in the records of Becker, Neal and Shealy, averaged 6,338 pounds of milk in eleven consecutive lactations and then broke her pelvis in three places as a result of depletion of bone on a calcium-low ration, but she did not show any sign of milk fever.

Temporary "disturbance of nervous endocrine equilibrium and disproportionate secretion of anterior pituitary hormones," as the precipitating cause of milk fever, remains the most attractive etiological hypothesis at present. Relapses may be speculatively explained as a second indiscretion on the part of an anterior pituitary fatigued in adjusting itself to the demands imposed by new lactation.

Hypomagnesemia and Hypomagnesemic Tetany

The terms "lactation tetany" and "grass tetany" are misnomers in the sense that there is no obligatory association with lactation or with grass. The "physiological status" may occur in stall-fed cows in winter or even in young bull calves on a predominantly milk diet—although it is more than possible that the common form of "milk tetany" in calves is an entirely independent condition in which low blood magnesium is an accidental concomitant.

It is true that in Holland the greatest clinical incidence of neuromuscular hyperirritability, as observed by practitioners, occurs at the time of turning out from winter stalls to spring grass but because of the exigencies of milk and cheese production in Holland this also happens to coincide with a "labile period" in the sexual life of the cow when she is sensitive to adverse influences of any sort.

It is true that in England more clinical cases occur over March, April and May than in all the other months of the year put together and that this period coincides with the flush of spring and early summer grass, but it is also true that there tends to be a recrudescence in autumn and that cases can occur at any time of the year even under stall conditions. Clinical attacks have been observed on Christmas Day in cows kept entirely indoors on concentrates and hay, but they are the exceptions deviating from the general "seasonal grazing rule" and only serve to warn against the idea that a "specific factor in young grass" is necessarily the cause of the disorder.

From a biochemical point of view, however, wide-spread hypomagnesemia, with normal blood calcium, may be found in mid-winter when grass growth is at a standstill, amongst gravid cows out all the year round and in good condition, although restricted to hay carried into the fields as the only supplement to grazing. Occasion-

ally a heifer or young male shows the same phenomenon, but data in this direction are scanty. There need be no symptoms of nervous hyperirritability whatever, even when the plasma magnesium is down to levels commonly found in acute convulsive tetany, and the low winter values may rise gradually to normal summer values right through the period of rapid growth of grass without a single clinical manifestation unless some very drastic precipitating cause upsets the unusual Ca/Mg ionic equilibrium to which the animals have successfully readjusted themselves. A common "drastic precipitant" is parturition at a time when the plasma magnesium is still low. The "downward parturient swing" of blood calcium, so common after normal calvings in overcivilized cows, and vacillation of blood magnesium, may be pathologically exaggerated to a dual hypocalcemic-hypomagnesemic state manifested clinically in any form from "atypical milk fever" to "convulsive tetany." The slight nervous hyperirritability so common in the early stages of ordinary milk fever may lead to convulsions instead of to coma.

The point is that this occurred on a large scale in 1937 in cows which would, without reasonable statistical doubt, have escaped the clinical syndrome entirely but for the coincidence in time between parturition and low seasonal plasma magnesium, *i.e.*, if they had calved two months later (as they did in 1936 and 1938 and 1939) no suspicion of any disorder would have been aroused in the mind of the attendant veterinary practitioner.

In cases of this type the ordinary calcium injection for milk fever is usually effective for the simple reason that if the calcium-regulating mechanism regains control the low magnesium is of less consequence because the animals have previously adjusted themselves to it. A simultaneous injection of magnesium, however, is all to the good in cases of threatened convulsions, even if only on pharmacological grounds.

The sort of biochemical picture to be expected in this type of case is 10mg% calcium and around 1mg% magnesium for some months before calving, figures such as 3mg% calcium and 0.6% magnesium at the time of attack, a normal recovery value for calcium but with the previous low magnesium of about 1mg slowly climbing back to normal summer value of about 2.3mg over a period of several months, just as it would have done if there had been no clinical attack.

That there is some sort of inter-relation between the physiological mechanisms which regulate the blood levels of calcium and magnesium and that the anterior pituitary plays a part in both, can hardly be doubted. Nevertheless, a great measure of independence is manifested. In the "seasonal hypomagnesemia without clinical symptoms" (recorded by Allcroft and Green) blood calcium need not be affected at all. Clinically acute hypomagnesemic tetany can occur with very rapid fall of blood magnesium, even to less than one-fifth its normal value and after recovery (magnesium injection) return to normal within a few days without material alteration of calcium throughout—indeed, in occasional cases, with a slight elevation of calcium to about 11mg%. On the other hand, the common type of case shows definite moderate concomitant fall of calcium —from 10mg down to about 7mg. In the cases best known to the practitioner, occurring at the time of rapid growth of pasture or after turning cows out from stalls to grass, the usual picture is a preliminary fall of blood magnesium over hours, days, or weeks, with latent tetany generally recognizable as nervous hyperirritability, but with no necessary change of blood calcium. Rough handling, a fright, sudden change of atmospheric temperature or undue disturbance of any kind, may then precipitate a sharper fall of magnesium, down even to one-tenth the normal value in extreme cases, accompanied by a moderate fall in calcium. The calcium may even fall to levels characteristic of milk fever but the disorder is clinically diagnosed as lactation tetany, partly because parturition has not been the precipitating cause and partly because the tetany induced by fall in magnesium overrides the coma which would follow low calcium.

Nevertheless the treatment for milk fever, calcium injection alone, is often effective in such cases, restoration of calcium control being followed by automatic readjustment to the "latent tetany level" of magnesium which some animals seem to be able to tolerate. A simultaneous magnesium injection, by alleviating the nervous hyperirritability, assists recovery and in some cases is absolutely necessary. The type of case in which a sudden fall of blood magnesium without material fall of calcium is manifested by acute tetany does not seem to respond to calcium but may show spectacular response to injection of magnesium sulphate, followed by rapid reassertion of normal physiological control. This is presumably the kind of case Dryerre (the first to follow up the Dutch work in this country and to establish the hypomagnesemic character of the equivalent English disorder) had in mind when he designed his magnesium lactate injection with omission of calcium.

In practice, however, since the veterinary surgeon cannot always distinguish which biochemical variation he is dealing with, it is best to use combined calcium and magnesium therapy. The unwanted ion appears to do no harm and in any case is rapidly distributed over the tissues so that any effect is evanescent. Indeed, in Holland, the "Sjollema injection," *i.e.*, mixed chlorides of calcium and magnesium, is almost invariably given, quite indifferently for "milk fever" and "grass tetany" on the grounds that a balanced injection reduces cardiac arhythmia. Since the chloride ion, in combination with the calcium ion, may induce local tissue necrosis, the injection is given intravenously with a special type of needle (double or short single) and a technique avoiding accidental subcutaneous delivery. In this country subcutaneous injections with calcium borogluconate and magnesium sulphate are preferred at different sites because of incompatibility of calcium and sulphate ions (formation of relatively insoluble calcium sulphate).

Results of therapeutic treatment are generally excellent all over Holland and in some practices in England, notably in Hereford, where J. R. Barker has had so much experience of the varieties of clinical manifestation of hypocalcemic-hypomagnesemic states that he can, in nine cases out of ten, forecast the results of blood analysis with almost uncanny accuracy. Barker's therapeutic results are almost as good as those of one Dutch practitioner who gets 200 cases of grass tetany a year with 100% recoveries. G. Webster, of Lancaster, has also generally had favorable responses, but in some practices magnesium therapy seems to be less effective and relapses are more frequent.

In this connection much seems to depend upon the rate of fall of blood magnesium prior to clinical attack. It is naturally difficult to get a sufficient series of pre-clinical blood samples, since the practitioner is not called in until the animal is visibly affected, but the following four main types seem to be distinguishable:

1. Fall of blood magnesium rapid; interval between latent tetany and convulsive attack short. Response to magnesium therapy spectacular and permanent.

2. Fall of magnesium slower; mild latent tetany presumably demonstrable for some weeks before precipitation of the first convulsive attack, which prompts the calling in of the practitioner. Immediate response to therapy often satisfactory but with tendency to revert to the latent tetany phase from which another attack may be precipitated by a variety of causes. This type may succumb during an acute convulsive attack but frequently passes through several slight attacks, making good spontaneous recoveries with blood magnesium slowly climbing back to normal in jerky fashion over several weeks.

3. Fall of blood magnesium so slow, over months, that the neuromuscular system has time to adjust its responses to the gradually changing Ca/Mg balance. No latent tetany at all and no convulsive attacks or other clinical manifestations, unless a fresh factor, such as parturition, induces an entirely new imbalance.

4. Fall of blood magnesium associated with fall of calcium within a few weeks of parturition. This "mixed disorder," atypical

milk fever or atypical tetany, seems to respond to calcium therapy with or without magnesium therapy.

With regard to etiology, there is little doubt that the underlying cause is inherent in the animal itself and associated with imperfectly coördinated endocrine function. The physiological data of the Italian workers, mentioned earlier, suggest a misfit between anterior pituitary and pineal glands, but the whole field badly needs further investigation.

As already mentioned, there is some sort of correlation between physiological control of calcium and magnesium blood levels. There is no doubt that in many cases of typical milk fever the plasma magnesium is doubled as the plasma calcium is halved —cases up to 6mg% magnesium have been observed in Mr. Messervy's practice in Jersey. There is no doubt that in most cases of tetany, falling magnesium brings lesser fall of calcium in its train, although the magnesium change has to be fairly rapid to have this effect.

But what is the cause of the endocrine dysfunction in hypomagnesemic tetany? Once the "labile state" is induced, a host of minor disturbances can precipitate the clinical condition, but what induces the "labile state"?

In the case of the physiologically analogous milk fever, occurring as it does at a time when a physiologically labile state is obvious, the mind has become accustomed to dodging the fundamental issue and there is a tendency to be satisfied with phrases such as "individual idiosyncrasy," "inherent individual weakness," "the modern dairy cow a pathological animal" and the like. The practitioner seems mainly worried by the fact that the disorder grows more protean in its manifestations and that "milk fever seems to be less easily curable than it used to be."

To some extent we have to look at "hypomagnesemic tetany" in the same light, however repugnant that may be to the enquiring mind. It may be largely individual and there is probably a genetic factor. The disorder is unknown amongst the low grade stock on poor mineral-deficient pastures in ranching countries such as South Africa (where it could not escape detection by the veterinary service) although well known amongst the better class of stock on the superior pastures of New Zealand. Grading up of stock combined with modern pasture management, particularly the use of nitrogenous fertilizer, has been associated with increased incidence in Holland, where the prevalence since 1927 has been pronounced. Such improved pastures are rich in protein, high in potassium and in nitrate at periods of rapid growth, and although these components are not regarded as the primary cause of tetany they are considered to be the "last straw that breaks the back" of cows already in a "physiologically labile state" because of "nervous endocrine imbalance" or "anterior pituitary overstrain."

In England the occurrence of hypo-magnesemic tetany sometimes seems to be associated with particular farms and not with others. Some observers, notably F. Blakemore, are very definite on this particular point and predicate a "positive causative factor in the grass of affected farms." The attempt to incriminate manganese failed. J. R. Barker, on the other hand, on the basis of extensive experience over a long period of time in the same practice, is of opinion that incidence is not localized—cases crop up indiscriminately and outbreaks may occur on farms previously free from the disease for many years.

Since the disorder has now been recorded from nearly every county in England local farm factors, where they exist, are not confined to particular soil types or geographical formations, but are found on chalk, clay, sand, peat and loam. Nor have they been reliably identified with any particular botanical flora; they occur on poor, rough pastures, on good, natural pastures, and on pastures specially seeded and heavily manured for maximum production of the best edible grasses.

Since constitution of herd and type of husbandry tend to remain fairly constant on particular farms, more than one cause, including genetic susceptibility, is generally in operation at the same time and it is difficult to disentangle specific factors by

any means short of long-range experiments in which progeny from one farm are interchanged with progeny from another. Such an experiment is contemplated in connection with an existing herd in Kent, upon which observations have now been in progress for two or three years.

In Holland outbreaks of foot-and-mouth disease have been followed by increased incidence of a type responding very poorly to therapeutic treatment. In England it has been observed that comparatively localized infections such as contagious abortion cause no change in blood magnesium but that when most of the organs are involved, as in the case of advanced generalized tuberculosis, there may be a distinct lowering of blood magnesium—almost as if the physiological controlling mechanism were somewhat affected. Such cases, however, are not necessarily more susceptible to convulsive attacks.

Sheep suffering from pregnancy toxemia but recovering after lambing have occasionally been found to show very sudden changes in blood magnesium (variations of 100% either way within four days), particularly if hypocalcemia has been shown at the time of lambing. Quite definite but not striking temporary hypomagnesemia associated with hypocalcemia has been observed in transit tetany of Welsh moorland ponies, disappearing within a few days. There seems little doubt that if blood samples had been procured in the Hyde Park cases of transit tetany in sheep, examined by Professor Wright a few years ago, both hypocalcemia and hypomagnesemia would have been detected—the symptoms and history warrant the deduction.

A curious feature of the incidence of bovine tetany in England is that there are "grass tetany years" and years in which the incidence throughout the country is almost negligible. The same holds for "seasonal variation of blood magnesium without symptoms." On the same farm with the same animals a group of cows (out all the year round) showed much more pronounced variation in 1937-38 than in 1938-39, with minima and maxima in different months. Are a high proportion of our English cows so "temperamentally labile" that they react to weather? On the same farm there was some evidence to suggest that the mere shelter from adverse weather provided by a belt of trees in one paddock and not in another, influenced the level of seasonal hypomagnesemia.

Sometimes the association seems to be with climatic variations encouraging sudden flushes of grass growth and periodic stuffing of the rumen. Is there a rumenal reflex stimulation or a "nutritional surfeit factor" which upsets a delicate endocrine balance in "labile" animals? Or are there mildly toxic factors in lush grass, especially on certain farms? Or do nutritional factors which stimulate milk flow, as fresh young grass is supposed to do, induce hormonic imbalance in a functionally imperfect anterior pituitary?

So many factors seem to play a part that much further observation is required to disentangle them. No single specific factor, overriding others, has been successfully incriminated as causative agent in hypomagnesemic tetany at any period of the sexual cycle, any more than any one single factor has been incriminated as creating susceptibility to the hypocalcemic coma of milk fever at the time of parturition.

Summary

A general biochemical essay concentrating attention on the balance in the blood between calcium, magnesium and phosphorus, with particular reference to endocrine control. The correlation between hypocalcemia, hypomagnesemia and hypophosphatemia, and nutritional deficiency of the three elements concerned is discussed in the light of knowledge of physiological mechanisms for controlling blood level.

Hypocalcemic coma (milk fever) is explained as a nervous endocrine disturbance in which hyperfunction of the anterior pituitary plays the most important part, rather than hypofunction of the parathyroids. Hypomagnesemic tetany (grass tetany) receives a similar explanation, also with incrimination of the anterior pituitary. The mixed hypocalcemic-hypomagnesemic disorder is also discussed.

Veterinary Practice in the Dominican Republic*

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THE Island of Santo Domingo lies between Cuba on the west and Puerto Rico on the east. The Dominican Republic occupies about three-fourths of the island —an area of about 50,000 square kilometers—the western fourth being occupied by the Republic of Haiti. The population of the Dominican Republic is 1,479,417, or 75.5 per square mile. Ciudad Trujillo, the capital and largest city, has a population of 40,000. The 1935 census report numbers the domestic animals as follows: horses, 265,881; donkeys, 137,268; mules, 44,528; cattle, 913,128; sheep, 34,828; goats, 373,204; swine, 852,973.

Having assumed the duties of Chief of Veterinary Service of the Republic only a year ago, I am not in a position to say definitely what diseases exist among animals on the island. But it appears that, with few exceptions, our troubles are similar to those of the southern United States.

Diseases of Horses, Mules, and Donkeys

Among the interesting diseases we have not seen elsewhere is epizoötic lymphangitis, called in Santo Domingo "Culebrilla" (little snake). While cases of this disease are not encountered daily, there are many within the Republic, and they seem to be increasing in prevalence. We saw four cases in a group of 20 horses recently. We know of no specific for it, but early extirpation is useful. Later, the free opening of the nodules and thorough cauterization by the actual cautery has proved valuable. In cases of large tumors, we are experimenting with neoarsphenamine, and also with soluble iodine combined with sodium iodide intravenously, but it is too early to report results.

Strangles is present to a limited extent.

Tetanus is rather common, but with treatment consisting of opening the infected wound, the use of antiseptics and 5000 unit doses of antitetanic serum, 25 to 50% of the cases recover.

The usual types of colics are few because of almost exclusive feeding on pasture or freshly-cut grass, Guinea and Para grass. On the other hand, a rather fatal type of colic is reported in certain sections. Unfortunately, we have not seen any cases of this, nor have we been able to perform an autopsy on one so far. The history given resembles very closely cases of colic due to larval strongyles which we saw in Puerto Rico and St. Croix.

Another condition, more common than in any other place we have been, is that of sore backs caused by improperly-fitting saddles. Perhaps this is due to the general use of the peculiar Dominican saddle.

Accidents and wounds are not uncommon. One interesting case occurred some months ago. A cow with a new calf being brought in from pasture by a man on horseback stuck her horn through the abdominal wall of the horse, just behind the last rib. When we arrived, at least two feet of small intestine was protruding. After thoroughly cleansing the loop and the surrounding hair with a solution of chlorazene, and enlarging the wound, the intestine was returned. The muscular walls of the wound were securely sutured with fairly heavy silk, and a few loose stitches were taken in the skin, so that daily washing of the muscular wound could be made.

When the muscular wound had healed nicely and the skin wound was closing up, three weeks after the accident, the horse

*Presented at a meeting of the Houston Veterinary Association, Houston, Texas, Feb. 4, 1937.

developed a typical case of tetanus. Five thousand units of serum were given via the jugular vein, and the next day 5000 units were injected around the wound. Ten days later the animal was in service again.

Bony excrescences are rare, though occasional spavins, ring bones, and side bones are seen.

Laminitis from road use occurs at times.

Horses, mules, and donkeys are seldom castrated. As a rule they give little trouble.

Occasionally a mare has a malpresentation of foal, but living "close to nature," this does not happen often.

We have had too little opportunity for investigation to say anything regarding parasites.

Diseases of Cattle

Cases of anthrax have been reported several times, but so far we have been unable to verify the reports.

Blackleg likewise has been reported, but not confirmed.

During the past year hemorrhage septicemia has been prevalent in almost all sections of the Republic. We were quite surprised when several farmers reported that they had many recoveries, following the administration of a double dose (10cc or more) of hemorrhagic septicemia agressin or bacterin. After losing several cases, we decided to try this treatment and were greatly surprised when even those cases showing blood in the urine made prompt recoveries. Only in far-advanced cases were we unsucessful.

The tuberculin test has revealed some cases of tuberculosis in imported cattle, but it is very rare; probably existing only in imported cattle.

No infectious abortion has been postively identified, but since a number of cattle have been imported from the United States, and abortions do occur, its existence is suspected and we believe it to be spreading. Discovery of retained placentas in cows fed exclusively on pasture strengthens this belief. If the membranes cannot be removed easily, we insert one or more uterine capsules, repeating every day or two until the membranes are expelled or can be removed without difficulty. When the attendant has already removed a part, we irrigate with acriflavine solution, then massage the uterus through the rectum from before backward, which causes straining, and lastly, inject a pint of uteroil. Occasionally, this treatment has to be repeated every day or two for a week or longer, but we have never had a case of metritis that did not respond to this treatment. After the uterus has contracted well, only massage is needed every day or two until no sign of the disease remains.

My predecessor, Dr. Rafaél Merens Montes of Cuba, diagnosed several cases of pseudorabies (Aujeszky's disease) in 1932. Since then no cases have been reported.

Keratitis contagiosum has been very prevalent in the past year.

Derrengue, posterior paralysis in young cattle, is frequently reported. Dr. Frank Hart, an American veterinarian who was here during the American occupation from October, 1920, to September, 1923, concluded from his experiments that derrengue was caused by a poisonous plant, one of the Zamias, but his conclusion has not been generally accepted. The name expresses only a symptom; the disease may have more than one cause.

Dr. G. A. Roberts, formerly of the North Carolina Experiment Station, has spent many years in the American Tropics. Ten years in Brazil, almost an equal period in the Virgin Islands, and shorter periods elsewhere in the Caribs have given him a knowledge of animal disease in the American tropics excelled by no American veterinarian. Throughout his long expatriation Doctor Roberts has made frequent visits to the United States and has kept in close touch with the march of veterinary medicine here.

Bovera or bobera, from bobo (fool), also known as pneumoenteritis, in calves, exists throughout the tropics and near-tropics. It is the most prevalent disease in the Island, and causes more fatalities than any other. In some grade dairy herds bovera kills 50% of the young calves from two to four months of age. It may occur in any month, and is far more prevalent some years than in others. It is apparently not an entity, but a group of infections not associated with gastrointestinal parasites. Many cases show *Anaplasma marginale* in the red corpuscles. No satisfactory treatment or control for bovera has been discovered.

Dr. G. A. Roberts and his home in Ciudad Trujillo

Piroplasmosis occurs only in newly-imported animals from tick-free countries. Out of 25 Angus and nine Holsteins imported the past year, none were lost from uncomplicated piroplasmosis, although three showed hemoglobinuria. We have found alternating doses of trypanblue with a soluble quinine, two to four grams, intraperitoneally, more effective than trypanblue alone. One three-year-old bull, after one week of high temperature, made a nice recovery.

Anaplasmosis, with visible symptoms, occurs in a variable proportion of imported animals. Of the nine Holsteins, three were treated for anaplasmosis. One was treated with sodium cacodylate and camphorated oil. Before death, its blood showed anaplasma organisms in 40% of the red corpuscles. The other two recovered after receiving plasmocacodine (Norden). One Angus heifer, after a high evening temperature, showed a subnormal temperature the following morning and died the next day of anaplasmosis. One two-year-old Angus bull and a heifer were treated with plasmocacodine after anaplasmæ were found in the blood. Two days later they showed hemoglobinuria and both anaplasma and piroplasma organisms in the blood. Both died.

Lung, stomach, hook, and nodular worms and liver flukes are rather common, but only on certain farms do they cause serious damage. The screw worm fly is of little consequence.

Diseases of Sheep, Goats, and Swine

Other than that they harbor parasites similar to those found in cattle, there is little to report about sheep and goats. The temperature of the tropics is not suitable for wool, so nature in time modifies the wool of the sheep to a short wool, resembling hair.

Hog cholera takes its toll here, but the writer has found a number of cases, diagnosed by the laity as hog cholera, to be something else. Swine erysipelas and necrotic enteritis are not uncommon.

Hydrocyanic Acid in Sorghum Silage

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ALTHOUGH many varieties of sorghum which may develop toxic quantities of hydrocyanic acid[1] are used in making silage, no reports of poisoning from this source have reached this Bureau. The question arises as to what becomes of the poisonous acid. It is known that shredded sorghum will develop hydrocyanic acid and that, if allowed to dry exposed to the atmosphere, a large proportion of the acid will disappear. Hydrocyanic acid is chemically a very active compound and can react with a large number of substances to produce compounds from which HCN cannot be reformed by any simple process. The absence of reports of silage poisoning implies that most of the toxic acid has disappeared by the time the silage is fed.

It has been stated[2] that "the poison is destroyed during the process of making" silage, so that ensiled sorghum is always safe to feed. Warth and Misra[3] fed sorghum silage to 18 calves for 12 weeks at the rate of two or three pounds, dry weight, per diem. No poisoning was observed. The HCN content of the silage is not stated. Acharya[4] reports that silaging sorghum reduces the HCN to negligible amounts. The ensilage was fed to cattle without untoward effects.

On the other hand, Boyd, *et al.*[5] found that ensiling Sudan grass did not lower the percentage of HCN with untreated grass or after addition of molasses. A slight decrease was noted with Sudan grass treated with hydrochloric or sulphuric acids as in the A.I.V. method.

No figures have been published for the HCN content of sorghum silage aside from those for Sudan grass referred to. It was therefore of interest to determine how the HCN content of the fresh plant and silage made from it might vary, and whether different varieties of sorghum might show differences.

■ Samples of 12 varieties were used. These were portions of lots being studied in connection with an investigation of seasonal and other influences on the HCN content of sorghum and were not especially selected. The plants were grown at the Bureau of Plant Industry Experiment Station at Arlington, Va. Collections were made each morning at 9 a.m., and the plants were carried immediately to the laboratory. The stalks were discarded and the leaves were minced through a food chopper. The product was thoroughly mixed and samples were weighed out for analysis. A quantity of this mixture was packed firmly into pint preserving jars so as to fill the jar completely. The rubber sealing ring was adjusted and the cover was clamped on. The weight of these samples varied between 393 and 438gm for different varieties. The jars were stored at 25°C. for four to four and one-half months and then the contents were analyzed. Three samples were found to have molded, two so seriously as to necessitate discarding them. On one the molding was superficial and the lower portions were found unspoiled. The molded layer was removed and the unspoiled portion used for analysis.

The pH of the first and last samples was taken by suspending 10gm of the silage in 100cc of distilled water and taking the conductivity of the mixture by the glass electrode. No. 86 gave pH 4.18 and No. 105, pH 4.11, somewhat lower than the optimum pH as reported by the writers[6].

[1] Couch, J. F., R. R. Briese and J. H. Martin, 1939. Hydrocyanic acid content of sorghum varieties. *Jnl. Wash. Acad. Sci.* 29, pp. 146-161.

[2] Anon., 1934. Poisoning of stock by sorghums. *Rhodesia Agric. Jnl.* 31, pp. 169-170.

[3] Warth, F. J., and Shari Kant Misra. 1927. Experiments on the feeding of sorghum silage and concentrate to scindi calves. *Mem. Dept. Agric. Ind.* 9, pp. 125-153.

[4] Acharya, C. Naracimba, 1933. Investigations on the development of prussic acid in cholam (*Sorghum vulgare*). *Ind. Jnl. Agric. Sci.* 3, pp. 851-869.

[5] Boyd, F. T., O. S. Aamodt, G. Bohstedt, and E. Truog, 1938. Sudan grass management for control of cyanide poisoning. *Jnl. Am. Soc. Agron.* 30, pp. 569-582.

[6] Couch, J. F., and R. R. Briese, 1939. Hydrogen ion concentration and cyanogenesis in sorghum. *Am. Jnl. Pharm.*, 111, pp. 55-64, 151-160, 193-201.

■ The analytical results are presented in Table I. In all but one case considerable HCN was lost during the ensiling process.

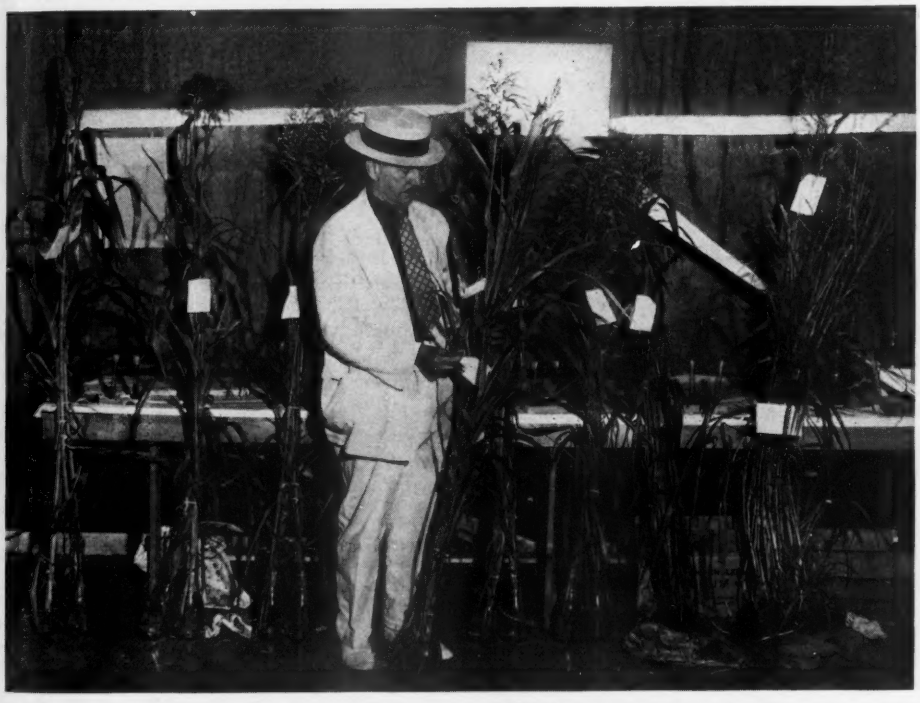
TABLE I. HYDROCYANIC ACID CONTENT OF FRESH AND ENSILED SORGHUM

Date 1938	Coll. No.	Variety	Time ensiled	Hydrocyanic acid in Plant Green	Plant Dry	Silage Green	Silage Dry	Loss
			Mos.	Mg%	Mg%	Mg%	Mg%	%
8/26	86	Spur feterita	4½	35	140	19.5	79	43.6
8/29	87	Hegari	4	33	141	26	111	21.3
8/30	89	Sumac sorgo	4	26	110	16	69	37.3
9/1	91	Grohoma	4	16	59	11	41	30.5
9/1	92	Ajax	4½	19	71	15	54	23.9
9/2	93	Milo	4½	11	40	7	27	32.5
9/2	94	Sagrain*	4½	11	43	6	23	46.5
9/7	96	Leoti sorgo	4½	9	35	9	35	None
9/7	97	Hegari	4½	35	140	21	84	40
9/8	98	Kans. Orange sorgo	4½	16	66	9	36	45.5
9/9	99	Rex	4½	9	29	7	22	24.1
9/12	102	Hegari	4½	19	88	13	59	32.9
9/13	103	Sharon kafir	4½	7	29	5	21	27.6
9/14	105	Atlas sorgo	4½	5	21	4.8	19	9.5

* Silage sample molded on surface.

The losses ranged from none for Leoti sorgo to 46.5% for the superficially molded sample of sagrain and 45.5% for Kansas Orange sorgo, the average loss being 29.7%. The fraction of HCN lost does not appear to be correlated with the quantity of potential HCN in the plant. Kansas Orange sorgo with 66mg% HCN in dry plant lost 45.5%, while hegari with 141mg% lost 21.3%, each losing 30 mg of HCN per hundred grams, but sagrain with 43mg% lost 46.5% or 20mg%. There was thus considerable loss of HCN but some samples still retained percentages of HCN usually regarded as above a toxic level.

Study of the rate of evolution of HCN in these samples showed that, unlike fresh plant, the whole of the available HCN was in a free condition in most of the samples. In one case, that of Kansas Orange sorgo



CYANOGENETIC PLANTS

Part of the exhibit of plants poisonous to livestock, at the clinic of the Omaha meeting of the A. V. M. A., Aug. 20, 1937. From the reader's left to right, the plants shown, are: (1) Black amber sorghum; (2) "Off type" plant found in black amber sorghum; (3) sudan grass-like plant, off type found in black amber sorghum; (4) sorghum-like sudan grass; (5) "off type" sudan grass; (6) normal sudan grass; (7) normal sudan grass, and (8) Johnson grass.

No. 98, the free HCN was but 50% of the potential quantity but in most other cases all or nearly all the HCN was present as such. This means that the full toxicity of the silage was immediately available and independent of any repressive factors in a digestive system. In the corresponding samples of fresh plant essentially all of the HCN was in the form of nitrile glucoside requiring hydrolysis before becoming free and toxic.

In working with these samples of silage it was observed that the HCN was rapidly lost upon exposure to air. In some cases more than 25% disappeared during 15 minutes exposure to the air. This suggests that, in feeding such silage, most of the toxicity is lost by exposure to the air before the animal ingests the material.

Summary

Samples of silage were made from 12 varieties of sorghum grown at Arlington, Virginia. At the end of four to four and one-half months the content of HCN was determined and compared with that of the fresh plant. In all but one case loss of HCN was noted in the ensiled samples, the losses averaging 29.7%. The loss was not correlated with the quantity of potential HCN in the plant. In the silage, except for one instance, all or nearly all of the HCN occurred as such, that is, as free HCN. In the fresh plant the contrary is true.

Some Pertinent Facts About the Soy Bean

In the long stream of agricultural history the soy bean has ever held a distinguished position. In America its rise in recent years has been phenomenal.

The soy bean, native of eastern Asia, is one of the oldest crops known to man. It was extensively grown long before written history began, some say 25,000 years ago. It was, however, long in finding the western world. Introduced into France as early as 1740 and in England in 1790, it was not until 1804 that the first soy bean came to America, and then as a curiosity cultivated only in botanical gardens. It was not until the 20th century that its commercial importance began to be suspected.

The first domestic production of soy bean oil in America was at Elizabeth, N. C., in 1915, in a cotton oil mill, using cotton oil presses. In 1920, soy bean oil was produced in a small flaxseed crushing plant at Chicago Heights, Ill., using both expeller and hydraulic equipment. In 1922, oil was made by a corn milling company in Decatur, Ill., and about 90,000 bushels of beans crushed that first year.

In 1922 only 99,000 bushels of beans were crushed in the United States. As recently as 1928-29, the industry produced only 21,000 tons of soy bean oil meal. Production of meal from the 1938-39 crop is expected to exceed 950,000 tons.

More than 95% and in some years as much as 98% of the soy bean oil meal produced in the United States is used in feeds for live stock, pet stock, and poultry.—*L. Kishlar.*

During the last 10 years, the amount of soy bean oil meal consumed by dairy cattle has increased rapidly. It has become one of the most popular protein concentrates, and is considered one of the most palatable protein feeds for dairy animals. In many comparative feeding tests, equal or superior milk production has been reported for cows fed this protein supplement. Considering average market prices over a long period of time, it has been a more economical source of protein for dairy feeds than other protein feeds of similar merit. —*J. W. Hayward.*

Hog feeders and breeders were among the first to recognize the value of soy bean oil meal. This is perhaps partly due to the fact that the beans are grown and the meal processed in near proximity to the hog growing sections of the country. However,

when we remember that until the advent of soy bean oil meal the hog feeders depended almost entirely on animal sources of protein even though several vegetable proteins were available, we must recognize that the real reason for this acceptance is the variety and balance of protein contained in soy bean oil meal. Soy beans themselves are not suitable for swine feed since they produce soft, and for this reason undesirable, pork.—*K. J. Seulke.*

There are at present approximately 200 brands of dog foods offered for retail sale. More than 500 million pounds are sold annually in this country.

Numerous nutritional feeding experiments have been conducted on the dog and other small animals in determining the best combination of food materials which should be used in preparing a high quality, well-balanced dog food. Perhaps the most important single nutrient to be considered in dog feeding is protein. Numerous laboratory experiments and practical feeding tests have been conducted to study the protein of soy beans. Raw soy beans were found to contain protein of low nutritive value, as determined by a unit of growth per unit of protein eaten. However, soy beans properly processed and at controlled temperatures were excellent in protein efficiency, superior to the proteins of other seeds, grains and their by-products and equal to some forms of animal protein. Dog food manufacturers, realizing the importance of the excellent source of protein in properly processed soy bean products, have used millions of pounds in canned or dry dog foods. It is a well-advised and usual practice to furnish the protein in a dog food primarily from meats, soy bean products, milk and grains. In addition to furnishing excellent protein, processed soy bean products contain appreciably more of the minerals, calcium and iron, than grain and grain by-products. However, when used to replace any large part of the animal proteins in dog foods, additions of calcium may be required to provide an optimum level of calcium. Processed soy bean products are also considered important sources of the vitamins belonging to the vitamin B complex. The widespread use of products derived from properly processed soy beans in dog foods of excellent reputation and proved high quality indicates that these products find important application in canine nutrition. —*H. E. Robinson.*

There are three methods for removing the oil from the soy beans in the manufacture of soy bean oil meal. They are the expeller process, the hydraulic process and the solvent process. Tests by recognized authorities show that soy bean meal produced by all three methods is satisfactory as a source of high quality protein in poultry feeding.

Unlike some commodities whose by-products have less food value than the original product from which they are made, soy bean oil meal has greater food value for poultry than the original beans. This apparent inconsistency may be traced to the fact that soy beans contain more oil than poultry can effectively use and also to the fact that heat treatment is necessary to bring about the maximum nutritive value of soy bean protein.

Research in recent years has shown that better results in poultry feeding arise when a variety of protein carriers, including both animal and vegetable sources, are utilized. It is rather well established that soy bean oil meal can be more effectively used in combinations with other materials such as milk products, meat by-products, high quality alfalfa and high-grade fish meal.

Soy bean oil meal contains an appreciable amount of several vitamins required by poultry. Its use is not advisable, however, except in combination with other vitamin-carrying ingredients.

This protein usually sells for less than do proteins from animal sources, and this lower cost, combined with high nutritive value, naturally makes it advantageous for the poultryman to use feeds in which a portion of the animal protein of the ration has been replaced with soy bean oil meal. —*J. E. Hunter.*

Bovine Verminous Pneumonia

By KENNETH L. JOHNSON,
Manhattan, Kansas
Kansas State College, Class of 1940

THE appearance of the lungworm *Dictyocaulus viviparus* in cattle, although perhaps not as common as lungworm infestation of sheep and swine, has been reported more frequently with each passing year.

As in other diseases, poor dietary conditions, unfavorable weather, intestinal parasites, and the invasion of numerous secondary organisms, often serve to mask the existence of the primary cause of the trouble. Therefore, the nature of the disease in an infected herd is not often recognized readily, and even in uncomplicated cases the animals may be treated for other conditions before the true nature of the malady is discerned.[1]

The male lungworm is 3.5cm to 4cm long. Its bursa and rays, the identifying portions of this white thread worm, are readily recognized. The female, 6cm to 8cm long, is ovoviparous. The paired uteri occupy almost the entire body and are filled with myriads of eggs containing larvae. The ova, 35μ by 85μ, are coughed up from the bronchioles, swallowed, and hatched in the intestinal tract. The larvae pass out with the feces; they are supplied with food granules so that they can develop in water. Under ideal conditions of temperature and moisture, they become infective in about six days, after undergoing two moults. In the infective stage, the larvae are very resistant.

Within 10 days after ingestion of larva-infected grass or pond water by a suitable host, the larvae penetrate the intestinal mucosa, moult a third time in the lymph glands of the viscera, and penetrate the bronchioles for a fourth moult. Development is complete in six weeks, so that the disease may be well under way before symptoms are noticed.[2]

The following case of lungworm infestation occurred at Arcola, Mississippi, in the Delta area several miles from the Mississippi River—a region with a warm, moist summer climate.

Late in July, 1939, the owner was pasturing approximately 400 head of mixed breeds of live stock in three pastures. Water was supplied abundantly in a number of troughs, but the cattle preferred to drink from a slough adjoining the pastures, where they were accustomed to stand during the heat of the day. This slough constituted a perfect incubator for the lungworm larvae, and samples of the water from it were found to contain large numbers of them.[3]

The major losses in this herd were among the young stock, the disease frequently affecting young cows just after parturition. In many cases the disease continued several weeks until secondary pneumonia or suffocation occurred. All of the young stock revealed some degree of infestation. Fecal smears showed a variable number of larvae, depending on the severity of the case.

The owner had entered the cattle business five years previously with fine pasture lands for summer feed supplemented by grain and alfalfa in the winter. During this five-year period stock was frequently added from Texas and the northern states. His first losses from lungworm infestation came in the fall of 1938, but were not severe. In the winter of 1938, losses were only occasional, but during the spring and summer of 1939, the severity of the infestation increased, and by the middle of August it was estimated that over 100 animals had succumbed, directly or indirectly, to this parasitism.

The entire herd of young stock had been treated with one or more liberal doses of anti-hemorrhagic septicemia bacterins.

[1] Pathology Exchange of the University of Illinois, 1938. Lungworm Infestation in Cattle. *Vet. Med.* 33:12, p. 575.

[2] See: Cameron, T. W. M., 1933. The Bursate Lungworms of Domesticated Animals (Imperial Bureau of Agricultural Parasitology); Hutyra, Franz, and Joseph Marek, 1938. Special Pathology and Therapeutics of the Diseases of Domestic Animals (3rd edition, Alexander Eger, Chicago), Vol. II, pp. 515-531.

[3] A similar case of stream infection has been described by Donald W. Baker: Some Common Parasitisms of Bovines Encountered in the Northeastern States. *Lederle Vet. Bull.* 8:3, pp. 41-46.

Mixed bacterins and anti-hemorrhagic septicemia serum had also been used.

All stages of the disease, with varying complications, were observed in the herd. A hospital ward was maintained at the barn lot, containing some 30 animals; new animals from the pastures were added daily.

Symptoms

A majority of the cases developed slowly, with a cough which became gradually more frequent and spasmodic. The animals became weak, anemic, and emaciated. Although they lost weight, their appetites remained fairly good.

In the chronic cases hospitalized, a dry, hacking cough was continuous. These animals stood in a depressed attitude, with their heads lowered; occasionally the tongue was protruded and masses of saliva drooled. The rate of respiration increased, and dyspnea was frequently present. Temperatures were seldom above 103°F. Diarrhea was invariably present. No discharge occurred from the eyes or nose except in cases which later developed into secondary pneumonia, with an accompanying rise of temperature to 106°F. or higher. The secondary invaders most frequently isolated from the lungs when so involved were *Pasteurella boviseptica* and Streptococci.

Most of the cases followed a chronic course, the animals continuing in a debilitated condition for a number of weeks until death from exhaustion or suffocation occurred. For a day or two before death, the animals lay on the ground with necks outstretched, gasping for breath. Hypostatic congestion in the lungs was a prominent feature on post-mortem examination.

Occasional "fall out" cases led the owner to suspect an acute febrile disease. Young, apparently healthy animals, after being driven, would suddenly drop, gasp for breath, and die with diffuse hemorrhages in the lungs.

Lesions

Unless lungworms are suspected, they are not readily found on post-mortem examination. An aid to their detection is the fact that when a piece of lung containing lungworms is allowed to remain at atmospheric temperature for several hours, the lungworms crawl into the larger bronchioles, where they are more easily seen.

Pathological changes in the lungs are not great in the case of uncomplicated verminous pneumonia. The lung is often emphysematous and pale, with wedge-shaped atelectatic areas, usually not apparent from the lung surface. The larger lung tubules contain a white, frothy material, which has a yellowish, mucous aspect in the smaller bronchioles, where cellular infiltration in response to the lungworm irritation occurs and a pneumonia develops. No secondary contaminating organisms are found in such lungs.

Young Stock Infested with Lungworms

Occasionally a gastroenteritis is observed, perhaps due to the irritation set up by penetration of the larvae, as suggested by Cameron and by Hutyra and Marek.[2] No other organs display any appreciable pathology.

Treatment

Prophylactic measures and good nursing are at present the only methods advised for combating lungworms. Since adult lungworms do not survive longer than 50 or 60 days, the animals free themselves of the parasites if reinfestation is prevented. The elimination of the slough was advised in this case, and severely affected animals were isolated and fed from racks uncontaminated with feces. Elimination of the disease from the herd will be a slow process, however, since although the slough is removed, the pasture itself will continue to harbor infective larvae. There is also the danger that neighboring pasture grounds will become infected, since the larvae are highly resistant.

Repulsion of Teeth in the Horse

By MERLE L. FARRIS, Nashville, Tenn.

Quite often in veterinary practice one is confronted with the problem of performing dental surgery in an attempt to correct a troublesome condition. In some instances the affected tooth may be simply cut off by means of the straight or compound molar cutters and the sharp edges floated, nothing more being necessary to remedy the defect. But in many cases the situation is not so simple as this and more painstaking treatment is required. When the condition is such that it necessitates repulsion, considerable skill and judgment must be exercised by the operator.

Indications

1. *Alveolar periostitis* is one of the most common dental diseases that is encountered in large animal practice. In herbivora the teeth are held in the alveoli by the alveolar periosteum or dental membrane with the outer layer of the membrane lining the alveolus and the inner layer covering the root of the tooth. At the connecting point between the periosteum and the jawbone the gum attaches and prevents the entrance of foreign material into the alveoli. According to Dollar there are two forms of this disease, (1) chronic ossifying and (2) purulent alveolar periostitis.

The chronic ossifying form is characterized by the formation at the root of the tooth of exostoses, which may vary from rough to flat, knot-like growths.

The purulent alveolar form is characterized by a thickened periosteum which is very vascular. At points it is covered with granulation and is separated from the wall of the tooth socket by a greyish-white, fetid, bone pus. Opposite these points the tooth may show signs of erosion to a considerable depth and in some cases there is evidence of suppurative osteomyelitis of the jaw. It may become so extensive that the tooth becomes loosened and can be lifted out with the fingers, but in most cases the operator must resort to trephining and repulsion.

Abscess Caused by a Diseased Tooth. This tooth, the first molar, was abnormally large; measuring 5½ inches from crown to root. An uneventful recovery followed extraction.

2. *Dental fistula* is characterized by a tube-like canal extending down through the infundibulum of the tooth, thus forming a communicating passage opening at one end to the outside and at the other end into the tooth, root cavity. It nearly always results from alveolar periostitis, which sets up an inflammatory process that produces a purulent osteomyelitis finally leading to perforation externally. When this inflammation abates, the alveolus alone remains inflamed and the small quantities of pus find exit through this fistulous tract. This condition is quite rare in the molars, but is more common in the premolars, especially of the lower jaw.

3. *Dental caries* is a term used to describe the diseased process which is a consequence of decay or destruction of the structural elements of the teeth. The enamel, due to its hardness, is very resistant and is destroyed much more slowly than are the other constituents of the tooth. Destruction of a tooth may result from any

one of several causes, but frequently it is due to a wearing away of the table surface down to the dentine or pulp cavity. Food particles penetrate the exposed cavity, decomposition takes place, and microörganisms propagate in it, with the result that the surrounding cement and dentine become carious. Gradually, the pulp cavity becomes open and is crammed with food particles which undergo putrefaction, producing a suppurative alveolar periostitis.

4. *Dental tumors* are seemingly quite rare, although there are a few cases of true odontomata mentioned in various texts. It is probable that most of the odontomata which have been mentioned in current literature are merely large exostoses which have developed on the teeth as a result of alveolar periostitis.

Instruments

1. Trephine.
2. Dental punch (may be straight or curved).
3. Rawhide mallet.
4. Mouth speculum.
5. Flashlight (built with bulb on end of 18-inch extension).
6. Molar extractor.
7. Scalpel.
8. Needles and syringe.

Technique

The animal is placed in lateral recumbency and properly restrained on an operating table, if one is available. If not, one must throw the animal and stretch it out on the ground between posts or stakes which have been driven in the ground as anchors. A speculum may then be applied and the mouth thoroughly examined to determine which teeth are involved. Too much stress cannot be placed on this part of the operation, inasmuch as the proper location of the trephine opening will determine largely the ease with which the tooth can be removed. It must be remembered that the transverse range will vary with the age of the animal, being well toward the median line in young animals and moving to a more lateral position with age. The longitudinal range will vary also, according to the tooth to be repulsed. The exact location is determined by passing the hand into the mouth and pressing a finger against the cheek opposite the affected tooth. From this bulge, a correct external measurement can be obtained. Then by applying one's anatomical knowledge of the positions of the roots of the various teeth, it is possible to trephine in a place that is most convenient to permit proper introduction of a punch.

The operative area is then cleaned, shaved, disinfected with alcoholic sublimate or tincture of iodine.

The nerves to the part are then blocked; if an upper tooth is affected, the infraorbital nerve must be blocked and if a lower, the mandibular alveolar.

A base line is next established. This is a line of maximum height where any superior cheek tooth can be repelled, extending from the medial canthus of the eye to just superior to the infraorbital foramen, and continuing in this general direction forward past the roots of the first teeth. It determines the position of the osseous lacrymal canal, which must be avoided in the operation. The trephine opening for all the superior teeth should be placed just inferior to this line except in aged animals, where it is permissible, though not necessary, to drop somewhat nearer the line formed by the facial crest, because the teeth are shorter. To locate the trephine opening for the first superior cheek tooth select a point below the base line directly above the center of the table surface of the tooth because this tooth is practically straight. For the 2nd, 3rd, 4th, and 5th teeth, select a point above the posterior border of the table surface of the teeth because of the backward curvature of the teeth. For the 6th superior cheek tooth trephine the frontal sinus on a line drawn between the medial canthus of the eyes and 1½ inches from the median line. Pass a curved punch through the trephined opening and through the frontomaxillary opening, carrying it lateral to the infraorbital canal to the root of the tooth.

In the repulsion of the inferior cheek teeth, the ventral lateral border of the mandible serves as a base line. The ventral border is chosen because the roots of the

teeth extend to the ventral border in all except old horses, and the narrowness of the mandible makes it necessary to have the punch absolutely in line with the long axis of the tooth in order to avoid punching toward and fracturing the medial plate of the mandible. The opening is made slightly laterally not because of the greater ease in working, but because the blood and nerve supply are located slightly ventral to the roots of the teeth. Consequently, in working through the ventral lateral border, one tends to avoid their destruction.

The trephine opening for the first inferior tooth should be located directly below the table surface of the tooth. The trephine opening for the 2nd, 3rd, 4th, and 5th teeth should be located directly below the posterior border on account of the backward curvature of the teeth. Repulsion of the 4th and 5th teeth is complicated by the presence of the external maxillary artery and vein and the parotid duct at the seat of the operation. To expose the bone before repelling the 4th tooth, make an incision medial to the artery and vein into the subcutaneous tissue and fascia around the border of the mandible, and draw them backward over the overlying structures. For the 5th tooth proceed in the same way at a point opposite the tooth, drawing the vessels forward. To locate the opening for the 6th inferior cheek tooth, draw a line from the center of the table surface to the point of greatest curvature of the mandible. Divide the skin and masseter muscle on this line and trephine the bone where palpation indicates that the two plates of bone are separated to accommodate the tooth. Due to the masseter muscle and its close insertion to the mandible, the incision should be liberal enough to allow retraction in order to expose the bone. It should terminate one to 1½ inches from the border of the mandible because of the location of the masseteric artery in this region. Be careful also not to divide the branches of the 7th nerve which spreads out over the surface of the masseter muscle from above downward and forward. The trephine opening should be elongated by the use of a chisel, thus allowing a better direction for the punch and preventing possible injury to the medial plate.

Treatment and Aftercare

After removal of the tooth, place a roll of bandage in the alveolar cavity and anchor it in by passing a string through the trephine opening and fastening it to another bandage roll on the outside. This prevents food particles from accumulating in the cavity.

It is well to remove the bandage roll from the cavity daily and irrigate the tract with some good antiseptic solution such as potassium permanganate.

Usually within six or seven days the cavity begins to fill in, and by about the 10th day healing is nearly complete.

Some use gutta-percha to pack the cavity instead of the bandage roll arrangement.

Possible Complications

1. Injury to adjoining healthy tissue.
2. Fracture of either the inner or outer plate of bone of the mandible.
3. In case the punch is placed medially, it will come in contact with the hard palate and may injure the palatine artery, causing profuse bleeding.

* * * *

Iliac Abscesses in the Horse

Iliac abscesses are located extraperitoneally within the abdominal cavity, at the anterior brim of the pelvic girdle along its supralateral arc, in the region of the iliac and sublumbar lymphatic glands and vessels. These abscesses may extend posteriorly into the pelvis in the periproctic region.

I have encountered a number of such cases; the majority following castration by laymen or quacks who used improper methods and disregarded antiseptic precautions. These abscesses occur also as a sequel to strangles, distemper, septic mammitis, septic wire cuts, and other injuries in the region of the iliac vasa afferentia.

Symptoms are usually overlooked in the early stages, or attributed to the infection already present. Later the animal will be reported as not doing well, losing flesh and

strength, lying down frequently, with unusual carefulness, rising as if in some pain, and suffering from difficult defecation and frequent micturition. As the abscess increases in size it causes more and more pressure on the rectum and floating colon and consequent obstruction, with corresponding symptoms. The abscess may rupture through the peritoneal wall and produce septic peritonitis, with its characteristic symptoms.

Diagnosis may be made readily by rectal exploration. The swelling is encountered at the location described; it might be mistaken for an impacted colon, but on manipulation it is found to be attached to the abdominal wall and along the ilium; it cannot be displaced, is tense, and does not pit on pressure.

Treatment must be directed toward draining the abscess exteriorly as soon as possible, in order to prevent its rupture into the peritoneal cavity, with almost certain fatal termination. I have found the following operative procedure the most satisfactory.

Restraint.—The animal may be secured on the operating table, in stocks, or with the casting harness, according to choice or facilities. General anesthesia is not necessary, since the operation is apparently not painful after the initial incision, for which a local anesestic may be used. I prefer to restrain the animal in stocks and induce general, incomplete narcosis with cannabis, administered intravenously.

Instruments.—A scalpel; a lance about two feet long (I made mine from heavy galvanized wire, flattened at one end and ground into the shape of a scalpel, with a loop at the other end so shaped as to indicate the facing of the cutting edge at the knife end); a long probe, gauze, carbolized oil (two parts of olive oil and one part phenol); and some lubricant (olive oil, vaseline, or liquid soap).

Technique.—Cleanse the field of operation, which is at the side of the anus corresponding to the location of the abscess. Make a vertical incision about two inches long. Lubricate the left hand and arm and pass them into the rectum. Taking the long lance in the right hand, pass it through the skin incision made with the scalpel forward along the course of the rectum to a point near the lower and external extremity of the abscess; carefully guiding the point of the lance with the fingers of the left hand through the wall of the rectum, in order not to injure the rectum nor the pelvis, nor puncture the peritoneum when the anterior part of the pelvis is reached. If the abscess extends into the pelvic cavity posterior to the pelvic reflection of the peritoneum, puncture its capsule. If the abscess is located anterior to the pelvic reflection, the wall of the pelvis is punctured with the lance and enlarged to an incision; this is accomplished by pressing on the lance with the left hand and drawing it gently back and forth with the right. Proceed with the lance in the direction of the abscess until the capsule is reached and punctured. An assistant may be required during the latter step to exert the necessary pressure on the patient's flank. Do not remove the lance when the abscess starts draining, but enlarge the entire artificial channel by gently pressing down on the cutting edge of the lance with the left hand and drawing it back and forth with the right hand, until free drainage is established. Empty the abscess by gentle pressure with the left hand. Saturate gauze with the carbolized oil and with a long probe pass one end through the operative channel into the abscess, directing it with the left hand as before, and leave the gauze *in situ*. The object of this procedure is to cauterize the channel, prevent healing by first intention and establish a route for drainage.

After-Care.—Leave the gauze in place for 24 to 48 hours, then remove it and irrigate the abscess with saline solution. (I use a horse catheter and water bottle for this purpose.) After irrigation, inject some mild antiseptic, such as borothymol solution, boracic acid solution, phemernite, or trypacrin. Repeat these injections every 24 or 48 hours, after emptying the abscess by gentle pressure with the hand through the walls of the rectum. If drainage is difficult, reinsert the gauze. Give internally tonics and alteratives such as Fowler's solution, iron chloride or neoprontosil. Supply laxative feed. Continue this treatment until the abscess is healed.

In my experience, the prognosis is far more favorable with this operation than with that by the inguinal route, where destruction of more tissue is necessary, and after-care is almost impossible. By the periproctic route there is a minimum of tissue destruction, and very little inconvenience during the after-treatment, which can be accomplished with the patient in a standing position by the mere application of a twitch.

J. R. ZECHA.

Ellinwood, Kans.

The Vitamin D Supply of Farm Animals

When animals are young and growing rapidly, vitamin D is especially needed for sound skeletal development. However, in many parts of the United States, young, growing animals may have little access to sunshine. Close housing is usually practiced in raising fall-farrowed pigs, as well as in raising "hot-house" lambs, which are born in the fall and must make their growth during the winter. In the case of "feeder" lambs, a report on practices in Western New York says that it is desirable to confine these animals during the winter, not only for protection from the elements, but also because a saving in feed is effected.

So far as calves are concerned, the rigors of the weather necessitate almost continuous confinement indoors during the winter in the northern states. This has been brought out by Bechdel and his associates, of the Pennsylvania State College, in connection with their study of the vitamin D requirement of calves. Moreover, calves are often kept housed for reasons other than protection against cold. As Morrison brings out, if calves are pastured at too early an age, there may be trouble from scours, and if turned out in the summer they may suffer from heat and flies. He goes on to say, "Many prefer not to turn calves on pasture until they are five to six months old, but prefer to keep them in the stable." Yearlings being prepared for show purposes are sometimes confined indoors, to protect them from flies and to maintain the appearance of the coat.

■ The amount of effective ultra-violet reaching the earth from the sun is markedly influenced by weather conditions, and fluctuates widely with the season. During cloudy and rainy weather, the vitamin D effect of sunlight is either eliminated entirely, or is greatly reduced. The effect of this upon the vitamin D supply is emphasized in a recent Illinois Extension Bulletin on rickets in calves, which states that, according to data of the Federal Weather Bureau at the University, during the four months of November, December, January and February, 1937-38, of 985 possible sunshine hours, 412 hours —over 40%—were cloudy.

Various investigators have measured the amount or intensity of solar ultraviolet at different seasons of the year. Dorno, at Davos, Switzerland, found that, in December and January, the intensity of solar ultraviolet at noon was only about one-twentieth as great as during July. Clark, in Baltimore, found, in two successive years, that the antirachitic irradiation from the sun at noon varied from about 500 ergs per square centimeter per second in December and January to about 6000 ergs in July. Tisdall and Brown, in Toronto, found that the average antirachitic value of sunshine in November, December and January was about one-eighth that during the period from April to August, inclusive. Somewhat similar findings were obtained at the New Jersey Experiment Station by Russell, Howard and Massengale, who used the acetone-methylene blue method.

The seasonal variations in solar ultraviolet disclosed by these observations are paralleled by observations upon the effectiveness of sunshine in the prevention of rickets. Day, at Little Rock, Arkansas, reported that for healing of rachitic rats an average of only five minutes of sunshine was required in May, June and July, as compared with 168 minutes in December. Rupel, Bohstedt and Hart, at the Wisconsin Experiment Station, in a study with calves employed a basal ration low in vitamin D, but supplemented with calcium carbonate. Calves which received this basal ration and winter sunlight showed lower serum phosphorus and poorer growth than calves on the same ration which were kept away from sunlight but received cod liver oil or ultraviolet irradiation.

■ It is known, of course, that sun-cured roughages contain vitamin D, but it is doubtful whether roughage can be depended upon at all times to supply an adequate amount of vitamin D when sunlight is not available, or when it is low in ultraviolet.

Sun-cured legume hays of good quality are, on the basis of available information, superior to other types of sun-cured roughage as sources of vitamin D. Many authorities on feeding, including Morrison, recommend that during the winter animals be liberally supplied with legume hay or with mixed hay high in legumes, as insurance against vitamin D deficiency. However, in practice, hay of this type is frequently not available. A survey during the winter of 1938-39 among dairy farmers in Kansas showed that only one-third of them used any alfalfa hay and that the roughage fed was mainly sorghum silage.

Even if legume hay is fed, it may be low in vitamin D if it has not been cured under favorable conditions. This was shown at the Wisconsin Experiment Station, by Steenbock, Hart, Elvehjem and Kletzien. They found that excessively weathered clover hay was much reduced in its antirachitic activity, as compared with hay which received less exposure to dew and rain during the process of curing.

■ In considering roughage as a source of vitamin D for farm animals, it is necessary to give attention to certain products which are being used to an increasing extent to replace sun-cured hay. One such product is "grass silage," which has many advantages. Harvesting can be done in weather unsuitable for making hay; on farms where erosion is a serious problem, legumes and grasses may be grown in place of corn; excess pasture grasses may be ensiled for use in the summer, when pastures are short. It appears that there is a sound basis for the growing popularity of "grass silage," and that it will be increasingly used, as indicated by the result of a recent survey. Of 380 Eastern and Middle Western farms, 224 reported that they regarded the ensiling of pasture crops as a basic procedure, and not merely as an emergency measure, when hay cannot be made satisfactorily.

This development is significant in connection with the vitamin D supply of farm animals. In making grass silage, the crops are ensiled almost as soon as cut, and therefore receive little exposure to sunshine. Fresh green plant material is essentially devoid of vitamin D. Hence, if silage is fed in place of sun-cured hay, there is a decided reduction in the vitamin D supply.

■ Another development to be considered is the use of artificially cured alfalfa. An

article in *Feedstuffs* for January 28, 1939, states that in recent years there has been an important increase in the production of alfalfa meal made from dehydrated alfalfa. This method of drying preserves much more of the carotene than does sun curing; however, it has been shown, that machine-cured hay contains much less vitamin D than sun-cured hay. Landsberg, Bechdel and their associates, at the Pennsylvania State College, found that 2½ pounds of sun-cured alfalfa protected one calf against rickets up to the time of slaughtering, at six months of age, but that on the same intake of artificially cured alfalfa, another calf developed rickets.

An article which also appeared in *Feedstuffs* calls attention to the decided reduction in the vitamin D content of rations which results when dehydrated alfalfa is used in place of sun-cured. The article states, "If dehydrated alfalfa is used, probably the inclusion of fish liver oil or a vitamin D concentrate in the winter supplement is advisable."

■ The amount of vitamin D obtained by farm animals is determined by the operation of various factors. These factors may combine to provide more than enough of the vitamin, or they may combine to bring about a borderline deficiency or even a serious deficiency, particularly in winter.—*Vitamin D Digest.*

The Avian Embryo
A Valuable Aid in Disease Study

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A SIGNIFICANT recent development in medical research is the utilization of the avian embryo in the study of certain phases of disease. While the incubating avian egg had long served as a fruitful subject for embryologic investigation, its potential value in the approach to various problems in other branches of biology, *e.g.*, bacteriology and pathology, was not recognized until the present decade.

As early as 1749 Beguelin made a window in the egg shell in order to observe the embryo during its development. Subsequently others employed the developing chicken egg for observation and experimentation upon developmental processes, tissue grafts and regeneration. The method of Peebles (1898) for manipulation of the embryo through a shell opening was applied by Rous and Murphy (1911, 1912, 1913) to the propagation of Rous sarcoma and other transmissible tumors in the embryo and its membranes. This work together with the injection of the egg with fowl spirillosis infection (Levaditi, 1906); with fowl pest virus (Juan and Staub, 1920); and with vaccine virus (Gay and Thompson, 1922) marked the first use of the egg in studying infectious agents.

It remained, however, for the excellent work of Goodpasture and his coworkers to reveal the possibilities of the egg in the attack on various disease problems. In their work with fowl pox, Woodruff and Goodpasture (1931) confirmed and extended previous observations that the embryo and its membranes were susceptible to certain virus infections and that the pox virus grew and multiplied enormously while producing characteristic lesions. Subsequently Goodpasture, Woodruff and Buddingh (1931, 1932) cultivated herpes simplex and vaccinia viruses in incubating eggs. Since then a great many viruses affecting birds as well as mammals have been grown and studied to advantage in eggs. Likewise, the introduction into incubating eggs of other disease agents including various bacteria, fungi, rickettsia and protozoa and their products have yielded interesting and valuable information.

These findings show that the embryos of various avian species are susceptible also to infection with viruses and other agents which in nature attack man and other mammals. The progress made by the use of the developing egg in the studies of pathogenicity, pathology and immunity is exemplified to a large extent in the work with the etiological agents of poultry diseases. To indicate the scope and possibilities of investigation in this direction it is, however, necessary to refer in several instances to the work with several mammalian infections.

Methods and Materials

The incubating chicken egg has been employed to a large extent for this work and found highly satisfactory. Low cost, convenient size, ready availability and relative freedom from natural and extraneous infection or contamination are major factors favoring the chicken egg over that of

other birds for the purposes desired. However, some of the comparative studies have revealed differences in susceptibility to infection and in the lesions produced in the embryo and extra-embryonic membranes of several species of birds. Differences in susceptibility and lesions have also been observed in eggs inoculated at different ages of incubation.

Provided with suitable conditions the fertile egg begins a remarkably rapid embryonic development. The onset of heart beat at about the 44th hour of incubation indicates how rapidly growth and differentiation processes occur. By the eighth day the extensive and highly vascular chorio-allantoic membrane has completely enveloped the embryo, its protective amniotic sac, and the yolk sac.

The fused chorio-allantoic membrane or serosa provides an actively functioning tissue which is particularly favorable for infection by a variety of viruses. Considerable manipulation of the serosa may be carried out without seriously jeopardizing the life of the embryo. With certain agents a benign and rather extensive function of the chorio-allantoic membrane may develop without involvement of the embryo and such an egg may hatch a normal chick.

The procedures in use for inoculating developing eggs are designed in general to permit introduction of infectious agents directly upon the serosa. The method originating in Goodpasture's laboratory consisted of removing a portion of the shell and underlying shell membranes from the side of the egg and, after the inoculum was introduced on the membrane, sealing the opening with petrolatum and a cover glass. Burnet modified this procedure to produce an artificial air sac, the base of which provides a relatively large area of serosa free from the bibulous shell membrane. Rivers and Schwentker (1932) in their work with Pacheco's parrot disease accomplished exposure by making a shell opening over the air sac and puncturing the shell membrane and underlying serosa with a virus-contaminated needle. The author (Brandly, 1935) introduced the inoculum "hypodermically" upon the serosa through a window over the air sac (see Fig. 1).

To facilitate injection into the embryonic tissues or the extra-embryonic sacs, various modifications of procedure have been de-

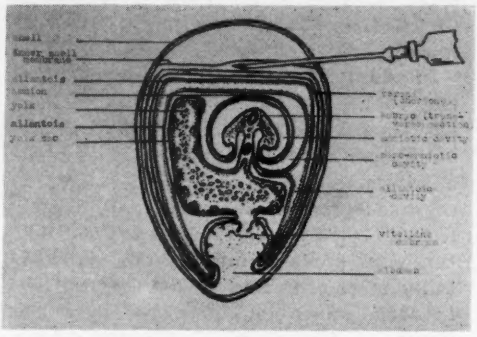

Fig. 1. Schematic drawing showing the extra-embryonic membranes of the chick and the point of inoculation upon the serosa (chorion). Ten-day embryo.

veloped. Dawson (1939) employed a method which allows satisfactory and uniform inoculation into the cerebrum of 12-day embryos. Inoculations of the vascular system of the serosa and of the amniotic and allantoic sacs may be accomplished also without serious sequellæ due to trauma.

After closure of the shell opening or window the injected eggs are subjected to further incubation. Certain effects on the membrane may be noted *in vivo* by later observation through the shell window, while candling of the egg reveals impaired development or death of the embryo. According to the object of study or use, the egg may be opened, examined and utilized at any desired time during the subsequent incubation period.

Many Viruses Cultivated in Eggs

While the major portion of the recent experimental work on eggs has dealt with viruses, some work on the pathogenesis, pathology and immunity of various bacterial infections has been reported. Goodpasture and Anderson (1936) in their studies on the problem of infection as presented by bacterial invasion of the chorio-allantoic membrane of chick embryos employed streptococci, staphylococci, *Br. abortus*, *E.*

typhi and *M. tuberculosis avium*. It is pointed out that the serosa provides the simplest tissue available for elucidation of the processes of infection and immunity. Other recent work deals with egg inoculation studies of *S. pullorum*, *H. influenzæ*, *H. pertussis* and certain fungi as well as spirochetes.

In addition to the virus of pox in chickens, canary and pigeon pox viruses have also been propagated continuously in incubating chicken eggs. Avian laryngotracheitis virus has been grown and studied on egg membranes and found quite rapidly lethal to the embryo. Strangely enough, this virus produces gross lesions and mortality in chicken and turkey eggs but not in duck, guinea fowl or pigeon eggs. The viruses of fowl plague, Newcastle disease, psittacosis, avian virus bronchitis, so-called equine encephalomyelitis and rabies have been found infective for developing eggs and passaged serially upon them.

Pathology

The lesions produced in the embryo and extra-embryonic membranes by many viruses show definite differential features, both gross and microscopic. Differences in membranal lesions among strains or variants of a certain virus (e.g. laryngotracheitis and pigeon pox) have been observed. In this connection, the value of the incubating egg is particularly apparent in that it may serve as a susceptible host or organism for comparative study of certain viruses or other agents which are innocuous for the usual laboratory animals.

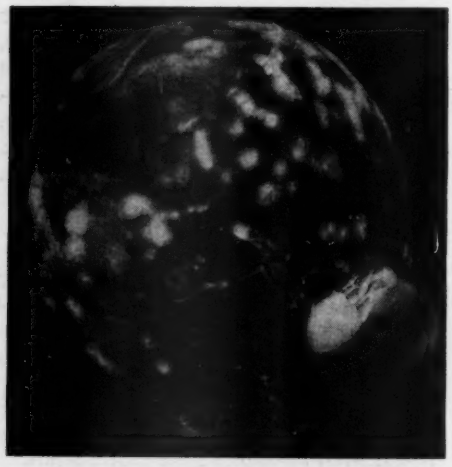

Fig. 2. Lesions of laryngotracheitis on chorio-allantoic membrane of a 17-day chicken embryo. This embryo was inoculated at 12 days of age.

Detection and Titration of Viruses

That the developing egg is a sensitive and superior medium for accurate titration as well as for detection of minute quantities of viruses has been demonstrated repeatedly. The tissue culture media in common use are largely derived and prepared from chicken embryos and such cultures *in vitro* have been found in several instances to be a more sensitive indicator of the presence of virus than the natural host or laboratory animal. Satisfactory titrations have been accomplished with viruses rapidly lethal for the embryo (fowl plague and equine encephalomyelitis) and also with others which produce plaques or pock-like foci of the serosa (the various poxes and infectious laryngotracheitis). The suitability of the egg for determining virus neutralization and as a means of serological differentiation between closely related viruses has been revealed by several workers. Burnet (1936) employed the chorio-allantoic membrane in elucidating the mechanism of the virus-serum reaction in laryngotracheitis.

Differences in pathogenicity and virulence of various bacteria, including *Pasteurella avicida* and several species of Salmonella, were demonstrated by inoculation of developing chicken eggs (Brandly and Graham, 1936). The titrations upon eggs were more accurate than upon experimental animals.

The sensitivity of the avian embryo as manifested towards viruses and other infectious agents has been demonstrated also with a variety of other factors and influences. Certain volatile agents (solvents) as well as mercury compounds, certain serums, snake venoms, various drugs and low temperatures elicit a sensitive response from the avian embryo, which may be put to practical usage. Elucidation of selenium

poisoning in poultry and other livestock was accomplished by injection of this element into the egg by Franke and coworkers (1936). The effect of the introduction of manganese into the albumen of eggs in the prevention of embryonic abnormality associated with perosis was shown by Lyons and Insko (1937).

Pure-Culture Viruses for Immunization

Because of several obvious advantages, pure culture, egg-propagated and tissue-cultured viruses have aroused much interest from the standpoint of immunization in poultry as well as in man and domestic animals. Vaccine virus propagated in eggs and in tissue-cultures has been used in human smallpox vaccination. In animals, egg-propagated and tissue-cultured fowl and pigeon pox viruses as well as laryngotracheitis virus grown in eggs have given satisfactory results in immunization of chickens against fowl pox and laryngotracheitis respectively. Almost invariably the concentration of the latter viruses in infected egg membranes is appreciably higher than in lesions from the chicken or pigeon. Another notable example of the greater abundance of virus in the embryo and its membranes as compared to that of the natural host tissues is seen with equine encephalomyelitis infection. Here the virus may be many thousand times more concentrated in the chick embryo than in the affected horse brain. This enormous multiplication is all the more remarkable in that it occurs within 12 to 24 hours following inoculation of the normal developing egg. The utilization of the virus from chick embryos for the production of a non-living "vaccine" for the successful immunization of the equine species as well as man is regarded as an outstanding current development in the virus field. The findings with various poxes and with encephalomyelitis virus carried for long periods through repeated serial passage indicate that in this process antigenic function and identity are not significantly impaired or altered. Certainly, the present status of virus disease immunization bids to be improved by further careful study and advantageous utilization of egg-propagated and tissue-cultured viruses.

Discussion

Only casual attention is necessary to impress one with the important part which the developing avian egg has played and may play again in future attacks on certain disease problems. That the potentialities of the method or methods are far from exhausted seems quite apparent. Certain limitations and pitfalls, however, must not be overlooked in the use of the incubating egg for the study of pathologic and related processes. Although the phenomena of invasion and infection of the embryo and the membranes may largely parallel those in growing and mature birds, certain anatomic and physiologic differences may impose definite limitations on comparative study. Failure to recognize that non-specific injury and embryonic mortality may be indistinguishable from that induced by specific factors may lead to confusion and error. The tendency to interpret alterations in the rapidly developing and often undifferentiated embryonic tissues on the basis of gross observation alone is an ever imminent hazard.

The possibility that undesirable characteristics or modifications of a virus may be brought out or established by propagation in eggs can never be disregarded, particularly from the standpoint of vaccine production.

It must not be overlooked that while the methods of injection and manipulation of the embryo are relatively simple, reasonable care is essential to minimize trauma and prevent bacterial contamination. A consideration of possible tissue affinity or predilection of the agent or substance injected will determine the route and location for introduction into the embryonic tissue or membranes. Rabies virus may be cited as an example of a neurotropic agent which apparently does not find the intact and innervated serosa a suitable site for establishing itself. Hence, cerebral introduction of this virus as practiced by Dawson (1939) assures immediate and successful contact with the central nervous system.

Summary

The results obtained in the utilization of the avian embryo in the study of various phases of disease emphasize the many uses and possibilities of the developing egg in this field. Among the advantages established for egg study are (1) propagation and study of various viruses in pure culture; (2) titration and detection even in minute quantities of infectious and toxic agents and antiserums; (3) pathologic and serologic study and differentiation of various infectious agents and their products; (4) elucidation of the role of certain nutritional and physiologic factors in disease causation; (5) utilization of egg-cultivated and tissue-cultured viruses in experimental and field immunization; (6) investigations of fundamentals of resistance and immunity; and (7) ready availability and economy of the developing egg as compared with the postnatal chick or usual laboratory animal, thus minimizing or eliminating the expense of quarters, equipment and feed; the hazards of cross and inapparent infections; and the dangers of dissemination of infection outside the laboratory.

. . . .

Milk Fever Complicated by Acetonemia

I was called to attend a grade shorthorn cow which had calved the day before. This was her third calf. The animal was in a semi-comatose condition, with a temperature of 99°F., and was lying in a typical milk fever attitude.

An intravenous injection of 300cc of calcium gluconate was made, and after the usual half-hour I allow for the injection to take effect, I got the animal onto her feet. She responded nicely, and I soon left for home feeling that we should have more specifics like calcium gluconate in our work.

The next night the owner telephoned to say that the cow was no better, having gone down again in about four hours after I had left. She had taken no feed or drink of any kind. Since this was a 25-mile trip, I postponed my visit until the next morning, when I found the cow in the same attitude as on my first call; the temperature was 99.5°F., and the animal was still comatose. I administered 250cc of calcium gluconate and inflated the udder. The bowels were absolutely devoid of action, so I pumped into her stomach four gallons of a saline solution along with two rumen compound tablets. In the course of the following half-hour the animal again got on her feet, and seemed interested in food. She was given a small bran mash. I left for home thinking that she would certainly be all right this time; but on the second day thereafter the owner telephoned to say that the cow had gone down again about six hours after I had left, but had wandered about in a small pasture, eating grass intermittently before being taken to the barn, where she went down again.

This time I took my acetonemia test equipment with me, finding the animal in the same condition as previously, except that bowel evacuation had taken place. The test for acetonemia was positive. I administered intravenously 500cc of 50% dextrose, and within a week the cow was well on the road to recovery.

The moral of this case is always to suspect acetonemia in cases of parturient paresis that do not recover promptly following injection of calcium gluconate, and to test for acetonemia immediately.

J. ARBUTHNOTT.

Stonewall, Manitoba.

Following the birth of an abnormally large calf, this Jersey cow's hind legs were paralyzed and the pelvic region swollen. The swelling subsided after drenches, but reappeared when bowel action was necessary. Although the cow's general health continued excellent and the calf flourished, the condition proved incurable.

Trichomoniasis in Turkeys

By L. D. BUSHNELL and M. J. TWIEHAUS, Manhattan, Kansas

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Introduction

NUMEROUS flagellated protozoa have been observed in the digestive tract of domesticated birds. Some appear to be harmless, while others are distinctly harmful. The one which appears to be of the most importance in this country is Trichomonas.

There is still much confusion concerning the classification of these parasites. Certain writers contend that there are different species present in the different birds and at different levels of the digestive tract of the same bird. We cannot answer these questions at present since the reports on experimental results are conflicting.

Parasites of this genus have long been recognized as causing infections in pigeons and the organism from these birds has been named *Trichomonas columbæ*.

Weinzirl[1] described a trichomonas infection in young chicks and named the organism *T. pullorum*. Jungherr[2, 3] was the first to describe the disease in turkeys. He first considered it to be a type of mycosis, due to a fungus. Later he was in doubt about the cause. Volkmar[4] found Trichomonas associated with the lesions in the crop and gave it the name *T. diversa*. He found this organism in all the organs of the digestive tract anterior to the ventriculus. Gierke and Hinshaw[5] described an intestinal type of the disease in which they observed a species of the parasite somewhat smaller than that seen in the upper levels. They were able to transmit the infection to young turkeys and chicks but not to older chicks. Gierke,[6] Vawter and Records,[7] Newsome,[8] Hawn,[9] and several others have described the disease and the parasite.

The death losses range from very few to as many as 73% for mature birds to 87% in young stock. Under field conditions in this area the disease is found more commonly in the anterior portion of the digestive tract in adults and in the posterior portion in young stock. There appears to be considerable evidence that there are two distinct forms of Trichomonas involved.

Etiology

Hinshaw[10] discussed the infection under two heads—intestinal trichomoniasis, of the lower intestine; and trichomoniasis of the upper digestive tract, including the organs from the mouth to the gizzard. He described the two different pathological conditions and thought them to be due to two types of the parasite. There is also some evidence that there may be cross-infection of turkeys and chickens with the pigeon type, *T. columbæ*. Stabler[11] maintained that he was able to transfer the pigeon parasite direct to the turkey, but without causing a pathological condition. The turkey remained a carrier and he was later able to reinfect the pigeon from the turkey. Twiehaus has been able to infect a pigeon with the turkey strain. The pigeon was still a carrier after nine months. Levine and Brandly[12] described a caseous lesion in the crop of the chicken which they considered as being caused by the pigeon strain.

Volkmar described the parasites of the upper digestive tract as 7.8 microns wide by 10.3 microns long. Hawn gives 9 by 14 microns as the average measurement. Hinshaw states that those from the lower intestine are somewhat smaller, with an average size of 4 by 9 microns.

In freshly prepared specimens the organisms exhibit a rapid motility due to the movement of the flagella. The number of flagella appears to be variable. The name

[1] Weinzirl, J., 1917. *Jour. Bact.* 2:441.
[2] Jungherr, E., 1927. *J. A. V. M. A.* 71:636.
[3] Jungherr, E., 1933. Storrs Agri. Exp. Sta. Bul. No. 188.
[4] Volkmar, F., 1930. *Jour. Parasitol.* 17:85.
[5] Gierke, A. G. and W. R. Hinshaw, 1936. *J. A. V. M. A.* 88:76.
[6] Gierke, A. G., 1933. Calif. State Dept. Agri. Mo. Bul. 22:205.
[7] Vawter, L. R. and S. Records, 1928. Nevada Exp. Sta. Bul. No. 61.
[8] Newsome, I. E., 1930. Colo. Agri. Exp. Sta. Bul. No. 372:31.
[9] Hawn, M. C., 1937. *Jour. Inf. Dis.* 61:184.
[10] Hinshaw, W. R., 1937. Calif. Agri. Exp. Sta. Bul. 613:80.
[11] Stabler, R. M., 1938. *J. A. V. M. A.* 93:33.
[12] Levine, N. D. and C. A. Brandly, 1939. *J. A. V. M. A.* 95:77.

would indicate three, and Volkmar reported three, Hawn and Stabler described four, and we have also noted four in this laboratory.

Gierke and Hinshaw described the disease of the posterior two-thirds of the intestine and considered that the parasite which they observed was similar to the one described by Hadley, *T. meleagridis*. Hawn considered that he was dealing with *T. diversa;* he could find no constant correlation between the presence of the parasite in the lower levels of the digestive tract and the presence of lesions.

From our experience it is difficult to correlate the clinical and pathological findings with the presence or absence of parasites. It is also difficult to classify the organisms on the basis of size, due to their great variation and to the fact that it is almost impossible to observe individuals that have not been distorted either by movements in the living forms or to fixation during the staining process. However, on the basis of age susceptibility and the location of the parasite there are indications that there are at least two forms. Whether the forms found in the turkey are the same as those observed in the pigeon has not been definitely determined.

Symptoms

Young poults appear drowsy and greatly depressed. They may stand or sit with eyes closed and allow their heads to hang with the beak resting on the ground. When handled the affected poult appears very active, but when allowed to remain undisturbed it soon assumes the position described. Other symptoms observed are sagging wings, loss of appetite, and emaciation. The droppings are usually watery in consistency and contain large numbers of the parasites. Young birds may die in 24 hours, while older birds may live for several days or even weeks. In older birds the crop area usually appears depressed or sunken and sometimes a pendulous crop is observed. The odor from the mouth of such birds is putrid and quite characteristic of this condition, but not diagnostic. Just prior to death there are repeated attempts at deglutition. When the birds die there is often an extension of the head and neck.

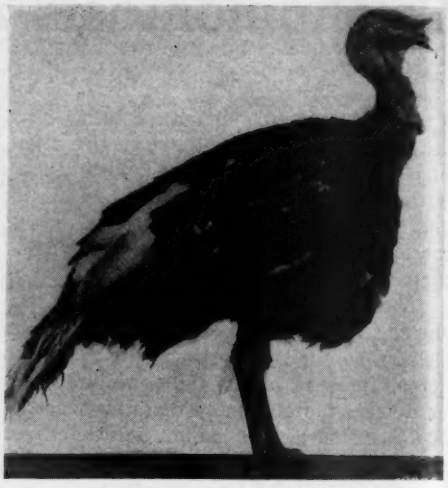

Fig. 1. This turkey exhibits the characteristic pendulous crop, with a depressed area above.

Post-Mortem Lesions

In the upper digestive tract the lesions appear as white or yellowish nodules located on the mucosa of the mouth, upper and lower esophagus and crop. They vary in size from 0.5 to several millimeters in diameter. In the crop the lesions are most characteristic. Here the infected mucous glands may contain a semi-caseous material and are surrounded by a ring of exudate. In the center of these areas there is frequently a thorn-shaped or conical elevation 1 to 3mm in height. These lesions may be so numerous as to form a confluent mass of necrotic ulcers over large areas of the crop wall (Fig. 2). The ulcers may penetrate the submucosa and occasionally may result in perforation. In acute cases an edematous infiltration may be observed in the tissues surrounding the crop. The areas of the crop which are not ulcerated do not show any significant changes.

In well advanced cases the mucosa of the proventriculus, especially of the anterior portions, may be necrotic and sloughing. It is usually covered with a thick, diffuse layer of semigelatinous mucus and exudes a sour, putrid odor. The gizzard is not involved. The crop contains a viscid, colorless or cloudy, watery mucous fluid filled with dirty white flakes, bacteria and flagellates.

The lesions in the lower intestine of

poults are catarrhal in nature. In early cases it is most pronounced in the anterior loop but gradually passes to the posterior areas. The ceca are frequently involved in an acute catarrhal inflammation. The flagellates may be found in myriads in these areas on microscopic examination.

The lower intestine and ceca are thin-walled and the entire intestine lacks tone. The contents are semifluid and contain gas bubbles.

Control

Hinshaw and Hawn recommend artificial incubation and brooding followed by isolation of the young birds in a clean environment. Adult and young birds should be separated since adult birds, although suffering no ill effects, may remain carriers for many months.

Stiles[13] states that prevention is more valuable than attempted cures. Isolation of sick birds, rotation of feeding grounds, clean water and strict sanitation are of value. He has suggested the following in the hope that some of these treatments may be of value:

Four parts of powdered catechu and one part of powdered ipecac. Give a heaping tablespoonful of the mixture in three gallons of water or in the feed. Continue treatment for about ten days.

Iodine, one part; iodide of potassium, two parts; water, 50 parts; heated at 158°F. with milk in proportion of one part of iodine mixture to nine parts of milk, until the mixture becomes white. Feed *ad libitum* to affected flocks in a dilution of one pint of the milk mixture to each gallon of drinking water.

Acriflavine in the strength of 1-5000 as reported for coccidiosis in rabbits.

From 5% to 10% dry sulphur in the mash for a few days as recommended for coccidiosis.

Copper sulphate in the drinking water, about one level teaspoonful to a gallon. Use several days at frequent intervals.

Calomel in one-quarter grain doses administered three times daily appears beneficial if begun early.

Several outbreaks of the disease in both adults and poults have been controlled near Manhattan by means of sanitation and the use of copper sulphate in the drinking water. Stiles recommended one teaspoonful per gallon. In our experience the birds will not drink a copper sulphate solution of this concentration unless starved for water for a long time and then they are sometimes severely injured by it. The use of one teaspoonful in five gallons of water is what we recommend. This gives approximately a 1-2000 solution. Birds soon learn to drink it so that it may be kept before them for several weeks as the only source of water without resulting in injury.

Fig. 2. Trichomoniasis, showing lesions typical of the disease in the crop and proventriculus.

In addition to the use of copper sulphate solution it is necessary to introduce sanitary measures, rotation of runs, and isolation of the birds from all outside contacts. One condition which appears to be especially important is to remove all wet and decomposing straw. The reason for this is not clear unless the parasite is protected against unfavorable effects of light and drying when the straw is present.

[13] Stiles, G. W. Rpt. Denver Office B. A. I.

The Large Roundworm of Chickens

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AMONG the important parasites of growing chickens is the large roundworm *Ascaridia galli* or *Ascaridia lineata*.[1] The habitat of the worms is the intestine, especially the portion anterior to the yolk sac diverticulum. The eggs, after incubating in the soil, hatch in the duodenum of the chicken. The larvæ move to a position a few centimeters posterior to the entrance of the bile ducts. Here the young worms live close to the villi for about ten days, after which they penetrate deeply between the villi, burying their anterior ends in the mucosa and destroying Lieberkühn glands. The larvæ remain in these positions for about a week, after which they withdraw into the lumen and move slightly farther back in the small intestine. Under favorable conditions they may grow to maturity in about a month.

Effects of Ascaridia on Chickens

The most harmful effects of the worms upon the hosts were found by Ackert and Herrick[2] to be in chickens from one to two months of age. When such chickens were infected with large numbers of the nematodes, serious results followed, especially during the second week of parasitism when the worms had penetrated the intestinal epithelium. Even though the experimental chickens were housed under the most favorable conditions of warmth and food rations, the mortality of the parasitized birds was more than five times that of the controls. In some experiments the mortality reached 67% of the parasitized chickens as compared with 3% in the controls.

From these and subsequent studies it was found that growing chickens, when heavily parasitized with this nematode, suffered from loss of blood and body fluids, reduced blood sugar, increased urates, shrunken thymus glands, retarded growth, and greatly increased mortality.

[1] These names, which are used widely in the literature, undoubtedly refer to the same parasite.

[2] Ackert, J. E. and C. A. Herrick, 1928. Effects of the Nematode, *Ascaridia lineata* (Schneider), on Growing Chickens. *Jour. Parasitol.* 15:1-13.

Resistance to Parasitism

Among the significant findings of Ackert and Herrick was the discovery of a potent age or natural resistance which reached its peak when the chickens were from three to four months of age. Later experiments showed that chickens raised under favorable conditions on an adequate diet developed sufficient age resistance in two and one-half to three months to largely protect them against the effects of the nematode.

These findings led to widespread efforts by poultrymen to keep the young chickens free from the nematodes until they were two to three months of age. Some poultry growers accomplished this end by raising the young birds over screen. Others used clean ground or rotation of runs, as findings from this laboratory at that time showed that the summer weather (sun, heat, desiccation) killed most Ascaridia eggs at or near the surface of unshaded soil. Cold also was found to be a lethal factor for eggs of the nematode. Repeated freezing at sub-zero (Fahrenheit) temperatures, or a single freezing at a temperature of —20°F. killed eggs on the surface of the soil.

It should be noted that the detrimental effects produced by infections of the Ascaridia were from rather heavy feedings of Ascaridia eggs to chickens one to two months of age. At the same time, however, the chickens were raised in a heated building and with the best ration known at the time. These conditions of rearing were much more favorable for the resistance of the host to the parasites than were those of the average farmer or poultryman. Not only was suitable temperature a factor but the ration likewise. Chickens then were frequently kept in runs which afforded no green feed and but little animal protein.

In order to ascertain what effect defi-

ciencies of green feed and certain other food accessories have, a number of experiments were conducted at the Kansas Station. Using rather large numbers of experimental chickens and control birds from the same hatches, tests were made on the effects of food deficiencies upon the resistance of the chickens to the Ascaridia with which they were parasitized. Tests from experiments showed that lack of vitamins A and B (complex) markedly lowered the resistance of the chickens to the worms. In the case of vitamin A the worms in the vitamin-deficient birds were both longer and more numerous than were those in the control birds to which vitamin A was supplied in abundance. Markedly more worms were found in the vitamin B-deficient fowls than in their controls.

Tests on vitamin D, as supplied by aërated cod-liver oil or with a mercury vapor quartz lamp, showed that while this vitamin did not increase the resistance of the chickens to the nematodes it protected the hosts against harmful effects of the parasite.

Another series of experiments gave evidence that milk is an important dietary supplement for high resistance to parasitism. Animal tissues in the form of meat scrap also constitute an important supplement to a plant diet. When a plant ration, consisting of a cereal-basal ration adequate for vitamins, minerals and salts was supplemented with meat meal and milk the chickens were much more resistant to the Ascaridia than were birds from the same hatch whose diet was wholly of plants, that is, food whose basal cereal ration was supplemented with peanut meal in place of meat scrap. Thus in order to develop in growing chickens a strong resistance against Ascaridia the ration should include a cereal-basal ration adequate for minerals and salts and be supplemented with suitable proportions of vitamins A and B (complex) and of animal tissues and milk (skimmed or powdered).

Among the factors necessary to produce a high degree of resistance in chickens to the Ascaridia is a normal amount of blood. When volumes of blood were withdrawn from parasitized chickens the resistance to the nematodes was reduced; the worms

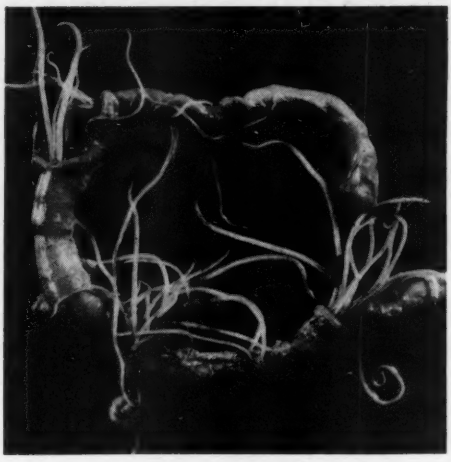


Fig. 1. Intestine of a six-month-old chicken suffering from nutritional deficiencies and heavy infestation of Ascaridia.

from such chickens were uniformly larger and more numerous than they were in the control chickens which had been given the same number of parasite eggs and kept under the same conditions except for bleeding. That the blood is an important factor in resistance is supported by recent observations of Clapham[3] who found that in cases of Leucemia domestic fowls had markedly larger numbers (198 to 247) of Ascaridia than did the fowls which had died from a cause other than leucemia. From the latter the *A. lineata* numbered from two to 37 worms.

The breed may be, to some extent, a factor in resistance to parasitism. Studies involving 1,351 chickens of five breeds and two varieties gave results which indicated that such heavy breeds as Rhode Island Reds and Plymouth Rocks are more resistant to the Ascaridia than are the lighter breeds of White Minorcas and White Leghorns. Even within a breed there may be resistant and susceptible strains of chickens. However, with the difficulties attendant upon inbreeding of chickens to main-

[3] Clapham, P. A., 1938. The Relation of Helminthiasis to Leucemia in Domestic Fowls. *Jour. Helminth.* 16:47-48.

tain the resistant strain the problem after a few generations becomes too involved to be practicable.

Control Measures

From the foregoing it is evident that many factors enter into successful raising of poultry. Within the flock, be it large or small, usually a few birds fail to develop normally. One or more of the important resistance factors fail to operate, with the result of the development, here and there, of a sickly fowl. Such birds are likely to become heavily infected with Ascaridia from failure to develop resistance against them (Fig. 1). The wise producer will isolate at once such inferior fowls.

Even in resistant chickens a few worms are able to mature and lay eggs to perpetuate the species; and since one female Ascaridia may produce as many as 100,000 fertile eggs, it is desirable to treat chickens for the removal of these worms. This should be done before the laying period as the egg production is materially reduced for a period of seven to 10 days following the administration of such an anthelmintic as carbon tetrachloride at a dose rate of 4cc per kilo of body weight. Treatment with this drug in individual gelatin capsules, inserted into the esophagus and forced into the crop, was found to be 100% effective on growing chickens, and practically free from toxic effects.[4] Other control measures involve adequate rations and suitable brooding and housing facilities so arranged as to keep the young birds free from Ascaridia until they are two and one-half to three months of age. After this time a normal, vigorous chicken may swallow many Ascaridia eggs without suffering noticeable effects.

[4] Ackert, J. E. and G. L. Graham, 1935. The Efficacy of Carbon Tetrachloride in Roundworm Control. *Poul. Sci.* 14:228-231.

An Itinerant Menace.—Drawing by Dr. J. R. Christian

Nicotine in the Control of Ascaridia Lineata in Fowls

By D. E. DAVIS, Petaluma, California

THERE are many poultry sections in the United States where *Ascaridia lineata* —the large roundworm of poultry—commands serious attention. It is reported that in some localities trouble arising as a result of roundworm infestation is not of great consequence, but there definitely are areas in which control of this parasite is a problem of considerable importance. If the manner in which intestinal parasites invade a poultry flock were spectacular, as is the case with some of the acute infectious diseases, poultrymen would immediately become alarmed and make some attempt at control. Parasites, however, like thieves in the night, creep unannounced into the flock and are very frequently well established and of long standing before poultrymen become aware of the fact that some hidden condition is seriously interfering with the well-being of their birds.

Sanitation, if it were properly carried out, would solve a number of our poultry difficulties, particularly those pertaining to parasites, but sanitation as it has been and will continue for some time to be practiced on the majority of commercial poultry farms is not a satisfactory solution to the poultry roundworm problem.

There are a number of drugs which might be satisfactorily used as a vermifuge but nicotine, from the standpoint of efficiency, availability (it is a common article of commerce), and low cost of treatment, appears to be the most practical drug yet found for the control of this parasite.

Physical Properties of Nicotine

The source of nicotine is tobacco. Tobacco, as it occurs in commerce, is of a yellowish-brown color, has a strong, narcotic, penetrating odor which is wanting in the fresh leaves, and a bitter, nauseous, and acrid taste. These properties are imparted to water and alcohol. They are injured by boiling, and the extract is, therefore, relatively feeble. The most important constituent is the liquid alkaloid, nicotine, discovered by Posselt and Reisman in 1828. Nicotine (Nicotina or Nicota), C_{10} H_{14} N_2, pyridyl-a-n-methyl-pyrrolidine, is a colorless, levorotatory fluid; specific gravity 1.010; boiling at 247° C. and not solidifying at —10° C.; having a faint odor when cold; of an exceedingly acrid, burning taste even when largely diluted; entirely volatilizable, and, in the state of vapor, very irritant to the nostrils, with an odor recalling that of tobacco; inflammable, very soluble in water, alcohol, ether, the fixed oils, and oil of turpentine; strongly alkaline in its reaction and capable of forming crystallizable salts with the acids.

Action of Nicotine

Nicotine primarily excites, and secondly paralyzes the ganglia upon the sympathetic nerves, stimulates the intestinal muscles, and, in sufficient quantities, has a paralytic action upon the motor nerves. As a result of action upon the sympathetic ganglia, it causes contraction of the blood-vessels, with a marked increase in blood pressure, followed, after large doses, by vascular dilation and fall of pressure. Pulse rate at first decreases, later becomes rapid. Nicotine acts very promptly upon the nervous structures in the body, particularly in those nerve endings which control various functions in the body relating to digestion, glandular action or muscular action. Large doses will produce, in general, a paralysis together with increased glandular activity with increased flow of secretion, and defecation.

Toxicity

In its action on the animal system, nicotine is one of the most virulent poisons known. A drop of it, in the stage of concentrated solution, is sufficient to destroy a dog, and five to 10 milligrams is a fatal dose for a chicken. Action is very prompt. There is no cumulative action. Recovery, if it occurs, is complete. It should always be borne in mind that nicotine is absorbed

readily by the skin and that serious and even fatal poisoning may result from its too free use and application on the surface of the body.

The fact that nicotine showed promise as a parasiticide along with the fact that it was inexpensive and so readily available in the form of tobacco prompted, in 1916, a series of investigations by Drs. J. R. Beach and S. B. Freeborn of the University of California on the value of tobacco infusion and tobacco dust in the control of *Ascaridia lineata* in fowls.[1]

Tobacco Infusion

The infusion was prepared by steeping chopped tobacco stems in water. One pound of tobacco (2% nicotine) was prepared for each 100 birds to be treated. The infusion was then added to a damp mash mixture and fed at once or added to a limited amount of grain and let stand over night. The soaked grain was then fed to birds which had been starved from 12 to 24 hours as the first feed of the day. This preparation was not readily consumed due to its unpalatability, as well as its extreme toxicity. The shock to laying hens was quite severe, causing a marked decrease in egg production, and frequently both young and old birds succumbed following treatment. This mixture, if consumed, would deliver approximately 90 milligrams of nicotine to each bird. The mixture consumed did, however, cause expulsion of numerous roundworms, indicating that nicotine would be a satisfactory vermifuge if the toxicity could be controlled and if birds could be induced to consume the feed.

Tobacco Dust Treatment

This treatment consisted of incorporating two pounds of 1% to 1½% finely-ground, dried tobacco into each 100 pounds of dry mash feed and keeping this mixture before the birds for a period of three to four weeks. Grain was also fed as usual. On the basis of 1,000 birds consuming 100 pounds of mash per day, the daily intake of nicotine per 1,000 was 10,350 milligrams of nicotine, or approximately 10.35 milligrams per bird. Although the birds did not seem to relish the mash there was no shock associated with this treatment. Some parasites were expelled, but due to the fact that the total dose of nicotine delivered was small, that it was delivered to the bird in small quantities throughout the day, and that some may have been absorbed before it reached that portion of the intestine inhabited by the parasites, the results were not all that was desired.

University Roundworm Capsules

In 1923 Dr. S. B. Freeborn, of the University of California Agricultural Experiment Station, conceived the idea of mixing nicotine with Lloyd's alkaloidal reagent and placing the mixture in capsules to be administered individually to birds. The formula, which became known as the University Capsule, was as follows:

6.6cc nicotine sulphate
16.0gm Lloyd's alkaloidal reagent
Mix thoroughly and place in No. 2 gelatine capsules, each capsule when filled to weigh 350 to 400 milligrams and contain approximately 46.6 milligrams of nicotine.

The introduction of this capsule marked the first time that large-scale individual treatment of fowls was attempted in California and the results demonstrated that individual handling was an efficient and practical method of administering medication to birds.

This capsule was a marked improvement over previous methods of treatment and for several years was generally accepted as the most efficient roundworm control available. There were certain factors relative to the University Capsule, however, which were not entirely satisfactory, and which, therefore, led to further investigation by other interested workers. The gelatine capsule which contained the University Capsule mixture was soluble in the secretions of the upper digestive tract and if not placed directly into the bird's crop would dissolve in the pharynx or esophagus, producing toxic symptoms and in many cases death. The capsule was also rather fragile and occasionally broke in the bird's mouth upon administration, causing death. It was advisable to feed birds before treating since

[1] California Agricultural Extension Service Circ. No. 8.

the capsule administered in an empty crop frequently produced toxic symptoms. Further, in the opinion of many poultrymen the dose of nicotine was not sufficient to produce the desired removal of the parasites. Autopsies of some of the treated birds revealed the presence of numerous roundworms, indicating that a more potent vermifuge was desirable.

Up to this time it had been demonstrated that nicotine would destroy the parasite, but no suitable carrier or holding agent had been developed with which it could be combined to render it nontoxic to birds when administered in a lethal dose.

In 1928 it was discovered that it is possible to mix a toxic alkaloidal substance (nicotine) in a sufficient quantity to eradicate internal parasites with an organic colloidal material and thereby obtain a mixture which is non-lethal to the animal to which it is administered even though the amount of the alkaloid would destroy the life of the animal unless held by an excess of the colloidal substance. Thus colloidally-held nicotine can be given without undue shock to a bird, even though many times the normally lethal dose is administered.

The writer's work in making a practical application of this problem, in which a considerable number of birds were studied, proved that satisfactory elimination of roundworms could be obtained by the administration of 70 to 80 milligrams of nicotine at a single dose. It has been repeatedly demonstrated that as high as 25 times the normal lethal dose of nicotine may be administered to a chicken when certain substances of colloidal nature are properly used as a holding compound. It is true that in cases of heavy infestation 70 to 80 milligrams of nicotine do not always cause the expulsion of 100% of the parasites but the percentage of removal is sufficiently high to make an increase in the dose, from a practical and economical viewpoint, appear unjustifiable.

Since it had been demonstrated that large doses of nicotine could be colloidally held, the next problem involved the release of the drug in the proper location in the intestine. In order to obtain a suitable parasiticidal effect, it is essential that substantially all of the nicotine be released at the location of the roundworms. The release must occur in sufficient amount and at a sufficient rate in the intestinal tract to kill the worms present and that rate must be maintained below the rate of absorption of the drug into the body so that it will not poison the bird.

It is possible in the mixing of colloidally-held nicotine to control the release of the alkaloid. It can, in fact, be made so the nicotine will be released in the upper digestive tract, in the alkaline portion of the intestine, or the nicotine can be so tightly held that it will not be released in a pH far in excess of the pH of the normal intestine.

In the case of a parasiticide for roundworms, the nicotine should not be liberated in the crop or gizzard of the bird but should be released by the alkaline digestive juices at the beginning of the second third of the small intestine. It is in this portion of the intestine, where certain secretions enter the intestinal tract, rendering the contents of the tract more alkaline, that the roundworms are normally found.

By applying the principle of colloidal holding, there has been made available to the poultry industry a parasiticide for the control of *Ascaridia lineata* which will deliver several times the normally lethal dose of nicotine without shock to young birds. It has no ill effect upon birds of laying age and consequently no detrimental effect upon egg production. It is insoluble in the secretions of the upper digestive tract but holds the nicotine in combination until it reaches the alkaline portion of the intestine. The nicotine is released at a rate sufficient to destroy the parasite but sufficiently below the rate of absorption of the drug into the body not to produce a toxic effect upon the bird. It is a stable compound resistant to moisture, temperature, and time.

This product can be made into a tablet for individual treatment, or in a granule form and mixed in a moist or dry mash feed for flock treatment. It is also inexpensive enough to be practical.

Paratyphoid Infections in Birds

By ERWIN JUNGHERR, Storrs, Connecticut
Professor of Animal Pathology, University of Connecticut

Introduction

BIRDS, in common with other animals and man, are highly susceptible to infection with paratyphoid organisms. The avian diseases caused by these organisms are usually of a septicemic nature, as are the corresponding diseases of mammals. In the latter group of animals secondary paratyphoid infections also occur, following initial biologic (virus) or physico-chemical injury, in contrast to man where the conditions are characterized primarily by gastroenteritic and toxemic (food poisoning) affections.

Paratyphoid infections are caused by bacterial organisms of the genus Salmonella (named in honor of Doctor Salmon of the Bureau of Animal Industry, who described the so-called "hog-cholera" bacillus, the type species of the genus). Since the generic name is often used in naming a disease group, paratyphoid infections are also known as salmonelloses. The bacteriologic concept of the genus Salmonella has been extended, however, by some investigators to include the causative organisms of pullorum disease (bacillary white diarrhea) and fowl typhoid—*Bacterium pullorum* and *Bacterium gallinarum**, respectively. Thus, while in modern veterinary-medical terminology paratyphoid signifies an infection with motile members of the genus Salmonella pathogenic for animals, the term salmonelloses comprises all infections engendered by the members of the genus Salmonella.

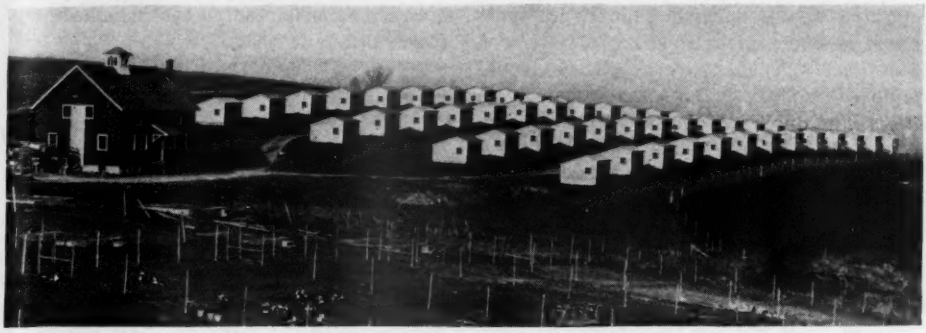
Recent studies of paratyphoid infections have been concerned with the accurate differentiation of the numerous species or types of the genus, especially on serologic bases, with a view to elucidating the epizoötiologic interrelationship of paratyphoid infections in various hosts. The widespread interest in paratyphoid infections in birds is due to the realization (1) that many serotypes of Salmonella identical with those in mammalian hosts occur in birds; (2) that a variety of avian species are subject to paratyphoid infections; (3) that birds may constitute an important natural reservoir of paratyphoid organisms; (4) that paratyphoid infections may occur in pullorum-clean stock and thus interfere with organized control programs for the latter disease; and (5) that paratyphoid infections of birds are of potential importance to public health.

*In the last edition of Bergey's "Manual of Determination Bacteriology" the former is designated *Salmonella pullorum* and the latter *Shigella gallinarum*.

Etiology

A brief statement is necessary here regarding the modern concept of the genus Salmonella, which is based upon accurate differentiation of serotypes. From the cultural and biochemical points of view paratyphoid organisms are defined as Gram-negative, motile, non-sporeforming organisms which ferment numerous carbohydrates with the formation of both acid and gas, but fail to attack lactose, sucrose, and salicin. Known partial cross-reactions among the various types, yet accompanied by definite serologic individualities in ordinary agglutination tests, have led to extensive studies of their antigenic composition, based on the following principles: (1) Motile organisms possess a group-specific heat-stabile antigen—the so-called body, somatic or O (abbreviation for German expression *Ohne Hauch*) antigen, and a type-specific heat-labile antigen, the so-called flagellar or H (abbreviation for *Mit Hauch*) antigen (Smith and Reagh, Weil and Felix); (2) The flagellar antigens may have either a specific group (monophasic) or may have both specific and non-specific groups (diphasic) (Andrewes). Both somatic and flagellar antigens consist often of several component factors which may be shared by other types in the genus. The antigenic factors can be recognized only by special agglutination tests with normal "smooth" representatives of the types (Arkwright).

Occasionally true paratyphoid organisms have been observed which deviate from the standard biochemical conception of the group (being lactose and sucrose-positive variants, etc.), a circumstance which led their geographic origin. Over 70 different serotypes have been recognized to date and some antigenic factors are known to be of greater diagnostic value than others. The system is necessarily in a state of flux, but



STORRS EGG LAYING CONTEST
This was the first international egg laying trial in the United States, established in 1911. The plant shown was rebuilt in 1935.—*Photo courtesy of Wm. F. Kirkpatrick.*

Kauffmann to define Salmonella as Gram-negative organisms which on the basis of antigenic structure can be fitted into the K-W scheme. Recent researches by P. R. Edwards of Kentucky point out that in some of the supposedly monophasic organisms the other phase(s) may be suppressed and is recoverable by appropriate technique. This would support "White's original view that the paratyphoid organisms as known today are loss variants of primitive types which had wide antigenic relationship and little or no host specificity" (Edwards).

On the basis of these fundamental conceptions of the antigenic structure of Salmonella, Bruce White developed an antigenic system which was extended and schematized by Fritz Kauffmann and is now known as the Kauffmann-White (K-W) scheme. In this system the principal somatic groups of Salmonella are designated by capital letters, the somatic antigens by Roman numerals, the specific flagellar antigens (phase 1) by small letters and the non-specific antigens (phase 2) by Arabic numerals, thereby allowing a mathematical formula-like expression of the antigenic structure of a given organism. The more recently recognized types are named after has furthered immeasurably the accurate identification of paratyphoid organisms, although it has also emphasized the complexity of the paratyphoid problem.

Occurrence

The occurrence of paratyphoid infection in birds is probably world-wide and extends over a wide range of avian hosts. Chickens, turkeys, ducks, geese, pigeons, pheasants, quail and canaries have been found to be affected, and among wild living birds, finches (*Fringilla*), siskins (*Carduelis*) and titmice (*Baeolophus*), and in zoölogic gardens gulls and parrots.

Edwards, in a recent report before the 1939 World's Poultry Congress, listed 16 different Salmonella types affecting birds in the United States, and Lerche three additional ones in Germany. All investigators agree that *S. typhi-murium*, formerly known as *S. aërtrycke*, *S. enteritidis* Breslau or the mutton type, is by far the most predominant organism (78%, according to Edwards); the Anatum type originally isolated by Rettger from ducks, has been found more recently in chickens and turkeys, and follows next in the order of frequency; but it, along with other rare types, is comparatively infrequent.

Failure to find *S. enteritidis* among the

types reported from the United States is noteworthy in view of the comparatively frequent occurrence of this type, together with some of its variants, in water fowl of Western Europe, especially geese and ducks.

Aside from *Salmonella typhi-murium* and *anatum*, the following Salmonella types have been observed in birds: *Amersfoort, Bareilly, Bredeney, Cholera-suis* var. Kunzendorf, *Derby, Enteritidis* var. essen, *Enteritidis* var. dublin, *Kentucky, London, Minnesota, Montevideo, Muenchen, New Brunswick, Newington, Newport, Oranienburg, Senftenberg*, and *Worthington*. These extensive typing studies have brought out the extremely interesting facts that some of the rare Salmonella types reported originally as associated with gastrointestinal disturbances in man in other countries, e. g. *S. bareilly* in India, occur in fowl in the United States; also that identical serotypes can be demonstrated in follow-up specimens from the same stock or the same premises after long periods, and thus become of epizoötiologic significance. The full import of these typing studies has not become clear because similar extensive serotype surveys are not available as yet for paratyphoid infections in mammals and in man.

Paratyphoid in birds is primarily a disease of the young. Epizoötics are often short-lived with some tendency for recurrence, but may become enzoötic. It is often difficult to trace the origin of the disease. In certain species of domesticated birds paratyphoid has become established and forms a well-defined disease entity.

In pigeons paratyphoid is probably of the greatest economic importance and is almost exclusively caused by a variant type of *S. typhi-murium* which has lost one of the heat-stabile antigenic factors (V) and can be subdivided on biochemical grounds, each type of which has epizoötiologic peculiarities (Edwards). The disease is often associated with trichomoniasis, another disease of squabs, and careful study is necessary to determine the etiologic factors involved. Similarly, in Europe ducks and geese are reported to be commonly infected by *S. enteritidis* var. essen.

Enzoötic cases of paratyphoid may constitute a danger to other birds and to mammals, such as rabbits (Bruner and Edwards), sheep and pigs. Flies and other insects act as mechanical carriers of the infection, but rodents, known to be very susceptible to paratyphoid infections, must always be considered as intermediaries between different hosts.

Symptoms and Lesions

In most species of birds paratyphoid infections are not accompanied by characteristic symptoms, with the exception of young pigeons, in which lameness and swollen joints are almost pathognomonic. Otherwise the symptoms resemble those of nonspecific diarrhea—droopiness, ruffled feathers, unthriftiness and increased mortality. Losses from the disease in young birds vary between wide limits depending upon sanitary conditions in the fly pens, the incubator and brooder, and upon the degree of infection in the parent stock, etc.

The lesions are likewise not characteristic, but ordinarily of sufficient intensity to suggest an infectious origin. Tumefaction and congestion of liver and spleen are quite constant. The hepatic changes may be accompanied by focal necrosis and distention of the gall bladder. Occasional entrance of the infection through the navel is suggested by an unhealed umbilicus in birds one week of age or older, by umbilical inflammation (omphalitis) and by persistence of a cheesy egg yolk. Inflammatory alterations are observed in the ceca (typhlitis) and on the serous membranes (peritonitis and pericarditis), but typical "pullorum nodules" in the epicardium and pulmonary tissue are exceptional. In addition, squabs are likely to show purulent arthritis, especially of the wing joints.

Older birds rarely suffer from the acute form of the disease, but may become carriers, with minor localized lesions. The ovary and testicles are predilective sites of infection and usually show some pathologic alterations; but, though similar in kind, they are rarely as extensive as in pullorum disease. This affinity of the infection to the gonads has suggested transmission of the disease via the egg, an assumption which

has been substantiated for several hosts, notably pigeons, turkeys, ducks and geese. Since the organisms localize frequently in the liver and intestine, paratyphoid organisms are eliminated by way of the feces.

The diagnosis should be established on the basis of isolation and identification of the causative organisms. Placing the isolated organism reliably into the genus Salmonella by biochemical and ordinary serologic tests is sufficient for most purposes, since serotyping is a procedure for specialized laboratories.

Public Health Importance

The sanitary importance of avian paratyphoid infections lies in the fact that birds now known to be a natural reservoir of paratyphoid organisms, are an important source of human food products. In Europe, human food poisonings have been traced frequently to infected eggs of ducks and geese, also of pigeons, and to infected meat products, especially goose liver and squab meat (Meyer). Probably due to the uncommon use of water fowl eggs and the rarity of avian *S. enteritidis* infections in this country, human food poisonings due to fowl products are extremely rare. Pigeon paratyphoid may be a potential source of danger to man, because it is widespread and squabs remain affected throughout the growing period. Luckily, affected squabs are also often thin and, therefore, unmarketable. Turkey paratyphoid seems to have made considerable progress during the past few years, but since it affects poults during the first weeks of life, the likelihood of infected birds reaching the market is not too great.

Obviously most concern should be felt over paratyphoid in chickens, since the consumption of chicken eggs is large and this often in a form which does not insure thorough cooking of the eggs to the point of sterilization. However, paratyphoid has not been demonstrated so far in adult fowl; in chicks of susceptible age it has been found only to the extent of slightly over 1% of the cases submitted to the Storrs laboratory. Although this leaves us in the dark as to the cycle of the infection in the common fowl, the actual danger from paratyphoid in chicks to human health seems to be slight.

Egg shells may also become contaminated by feces and thus constitute a danger in handling food products.

Very little is known as to the direct transmission of animal paratyphoid to man. What appeared to be an accidental laboratory infection in man by an avian strain was observed in the author's laboratory. The assumption that it occurs rests upon the findings of identical serotypes in man and animals. Further epidemiologic studies of human paratyphoid as to serotypes involved will shed light upon the true relationship of avian paratyphoid to corresponding human ailments.

Control

The control of paratyphoid infections in birds must be viewed from two standpoints, that of man and that of animals.

To avoid food poisoning, it has been a practice for several years in Western Europe to label duck and goose eggs very plainly and to request thorough cooking of such eggs. Although some paratyphoid organisms have been credited with a thermostable toxin, this practice seems to have been effective, probably due to the fact that heat sterilization inhibits the multiplication of organisms in poorly refrigerated food substances. Somewhat more difficult is the control of paratyphoid food poisoning from meat products, but again proper refrigeration at the time of preparation, followed by heat sterilization, will go a long way. Otherwise, general sanitary principles must be relied upon in preventing the cross transmission of animal paratyphoid to man, accompanied perhaps by a slaughter policy directed against known paratyphoid foci in animals.

Animal paratyphoid infections are admittedly difficult to control. Strict separate maintenance of every species of domesticated animal on the premises is one of the cardinal principles. The importance of rodent control must not be underestimated.

Specific methods which have been applied to the control of paratyphoid infection in birds are vaccination and eradication.

Some favorable results with formalinized

autogenous bacterins have been reported in the control of paratyphoid of pigeons in Roumania. Since the disease affects young birds before a partial active immunity can be set up, not much can be expected from this method. Furthermore, it should be kept in mind that vaccination would interfere with a blood testing program.

Attempts have been made to detect infected animals by serologic tests and by cultural examination of the feces. In serologic tests the number of etiologic types involved in avian paratyphoid demands the use of autogenous strains, preferably in the form of fresh live antigens. Most of this work has been done on pigeons. While it is known that repeated serologic tests of the parent stock followed by elimination of reactors will increase the viability and marketability of squabs, actual eradication is difficult to accomplish. Furthermore, the correlation between serologic and cultural reactors is much lower than in pullorum disease in which a similar program is found eminently successful.

Ordinary serologic tests for pullorum disease carriers will detect a certain percentage of paratyphoid carriers, but cannot be relied upon as an eradication measure for paratyphoid.

Cultural examination of the feces for the detection of paratyphoid shedders has also been tried. Aside from the amount of routine work involved, this procedure likewise is not too promising, since instances are on record in Western Europe where duck eggs from parent stock, negative to both serologic and fecal-cultural methods, have given rise to food poisoning in man. This would suggest that either not all infected animals were detected or that reinfection occurred from other sources.

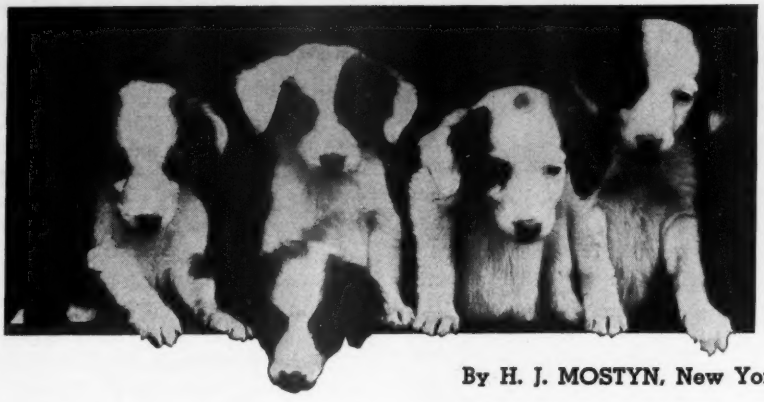
Chemotherapy, especially with sulfanilamide, has been unsuccessful in the few instances reported.

Sanitation at time of hatching, either under natural or artificial conditions, is probably one of the most potent factors in the control of avian paratyphoid. The fact that the organisms frequently find an avenue of entry through the navel suggests this. Attention should be paid to formalinization of incubator equipment, shipping boxes and packing rooms, and last but not least, to sanitary supervision of the persons engaged in these operations.

INTUSSUSCEPTION IN THE OVIDUCT OF A HEN

Intussusception in the oviduct of a hen. 1. Suspensory ligaments. 2. Oviduct. 3. Point at which intussusception occurred. 4. Oviduct. Along the middle portion of the oviduct (between 2 and 3) was a soft enlargement; the intussusception was just anterior to this.—*M. A. Schooley.*

Enemata in the Dog



By H. J. MOSTYN, New York City

THE origin of the "enema" is lost in antiquity. Hippocrates, "father of medicine," preferred this method of relieving costiveness, and we may be sure also that he used relatively simple formulæ, for one definite mark of an unethical practitioner in bygone centuries was a complicated enema prescription. Aetius, a well known Greek of the sixth century, used water; Galen, oil and honey; and de Graf recommended sea water or plain water with salt or urine in it.

Herodotus, the Greek historian and keen student of Egyptian customs of his day, states that the Egyptians purged themselves every cycle of the moon "thinking thus to maintain a state of health by the use of emetics and clysters, because it is their belief that all disease has its origin in disagreement of food eaten."

Regnier de Graf, in his 17th century treatise on the enema, cites the legend of the origin of the enema in this fashion: "Not to man belongs the glory of having invented the clyster. Rather should it be accorded to animals—acting under the guidance of their natural instincts. It was in Egypt that a bird, somewhat resembling our swan, called the ibis, first taught humankind the use of the clyster. When this bird found himself inconvenienced by an excess of painful humors, he filled his beak with sea water and introduced the fluid into the intestine, when it poured out abundantly, eventually carrying with it the burden which had incommoded him." This sounds fascinating, but unfortunately the Egyptian observer was not acquainted with the fact that the ibis has an oil gland adjacent to the anus, and in dressing his plumage he first fills his bill with water and then lubricates it by pressing it against his oil gland. The Egyptian student of bird life, watching this maneuver, must have been greatly amazed when the urge to defecate struck this ibis immediately afterward. The two acts were seemingly related. The deception was made even more complete by the fact that the ibis evacuates both urine and feces through a common cloaca, accounting for the enema-like appearance of the stool. The human dwellers along the Nile practiced what they thought the ibis had taught.

So down through the ages has come the idea of the enema. Although superseded by modern equipment, de Graf's syringe was a credit to the 17th century and an advanced instrument as compared with some of the more primitive instruments, which are still used among natives in certain sections of India and a few African tribes. A cow's horn, hollowed out and the small end pierced, is inserted into the anus while the patient sits in a swiftly moving stream, his

back toward the direction of the current. The horn is held in place until the water thus forced into the rectum produces a sensation of fullness of the lower bowel. The horn is then withdrawn, the bowel content expelled, and the procedure repeated until relief is obtained. A similar method employed among some of the desert tribes of Arabs, where swiftly running streams are not to be found, calls for a cow's horn inserted into the anus in the same manner, but the water is poured into the large end by a second person.

De Graf introduced a flexible rubber hose between the syringe and the tip, so his idea in this respect is still being carried on by modern manufacturers of enema equipment. Leroy d'Etioles earns the credit of changing the syringe type to the hanging receptacle form we now know. This was after the days of Louis XIV and Napoleon. The syringe type of enema was the vogue of the French court during the reign of Louis XIV. So great was the rage for rectal washing that wits dubbed this period in French history as "the age of the clyster and the periwig." The nobility were served by the court physicians by syringes whose nozzles were of gold. Even the fact that feminine modesty did not prevent the ladies of the court from receiving a daily internal bath is conclusive evidence of how the fad was followed. It was not until late in the 18th century that the enema came to be regarded as a therapeutic measure instead of a fashion.

The first case of rectal feeding is credited to the famed Moorish physician, Avenzoar (1113-1162) who struck a modern note by prescribing nourishment by way of the rectum, after a cleansing enema, in cases of paralysis of the pharynx.

■ Colonic irrigation can be defined as lavage of the large bowel. It is looked upon by many physicians as a "glorified enema." Bastedo[1] stated that "while the ordinary enema is given for the purpose of inducing defecation, the irrigation is administered, not to induce defecation but to wash out material situated above the defecation area and to lavage the wall of the bowel as high as the water can be made to reach."

Those who employ colonic irrigation do not place it in the same category as the enema, for the procedure is usually to empty the bowel by means of a purgative or enema prior to colonic irrigation.

It is extremely difficult to determine the exact sphere of its usefulness. Rankin, Bargen and Brire[2] state: "We have judiciously avoided comment on the use of colonic irrigation. Our experience would diminish its use." Indeed, as Friedenwald and Feldman pointed out in 1931, prolonged use of even the simplest enemas is irritating to the colon of dogs. Tidy has suggested that "medicated" enemas continued over a period of time would induce colitis.

P. W. Brown in 1933 wrote, in discussing diarrhea: "Irrigation of the colon has little if any place in the treatment of diarrhea. The occasional use of a small warm saline enema may be of aid, but its constant usage is not advisable."

There is nothing to be gained by the insertion of many inches of rubber tubing, except for the possible affect it may produce upon the owner of the animal, for the opaque enema in roentgen work has demonstrated beyond question that with a tube inserted three or four inches into the rectum, and a pressure level of two feet, the liquid will reach the cecum in two to five minutes. There is no specific difference, therefore, between a colonic irrigation and the ordinary enema.

Roentgen plates have also demonstrated the fact that in man the ordinary colon tube coils up in the upper rectum if the tube is inserted beyond six inches. There is no benefit to be derived from passing a tube 18 to 30 inches to form a coil. The use of copious amounts of fluid is also open to criticism.

Colonic therapy in human practice has been a "racket." Charlatans engaged in giving internal baths try to outdo one another in concocting new and "better" solu-

[1] *J. A. M. A.*, 98, pp. 734-36.

[2] Rankin, Bargen and Brire, 1932. "The Colon, Rectum and Anus" (W. B. Saunders Co., Philadelphia), p. 328.

tions to be used in irrigating the colon. Many of these patented solutions occupy a position today on the shelves of "modern" pet shops. They are usually either mild antiseptics or alkalinizing or acidifying solutions.

Since altering the reaction of the contents of the colon is apparently a futile procedure, as has been shown in many experiments, any attempt to sterilize the intestine by ordinary antiseptics is simply waste effort. Ordinary physiologic saline solution at body temperature is probably more satisfactory than "antiseptic" solutions. The use of very hot solutions to destroy intestinal parasites is of very doubtful value. Any intestinal parasites recovered from the feces by this method are merely indicative of the extent of infestation.

■ There are many elaborate devices for the administration of colonic irrigation. Super salesmanship has resulted in these being "adopted" by some human hospitals, so it is small wonder that some may be found in small animal hospitals whose patients are in the upper strata of dogdom. Some practitioners who succumbed to temptation and added these attractive looking apparatuses to their equipment have realized that the purchase price could have been spent in many better ways. If colonic irrigations are to be given at all the work can be done easily with an ordinary table, a plain glass jar on a stand, a rectal tube and a Y tube with two clamps, and a large jar to receive the return flow. The receiving jar is used only when it is suspected that the bowel content may be of diagnostic value.

For any colonic lavage, it is safer to use a velvet-eyed tube with closed end than one of hard rubber with a sharp-edged terminal opening.

In man, the possible usefulness of irrigation is limited to the treatment of focal infection in the colon or to the removal of impacted or pocketed fecal masses. Observation would tend to minimize the contention that the colon is often a focus of infection but it still remains controversial. Proper diet will correct a chronic constipation and obviate the necessity of using irrigations to remove fecal matter. Certainly oft-repeated irrigations are an

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abnormal procedure and to be strongly deprecated. At the most even occasional irrigations in man are rarely indicated, if we may accept a consensus of opinion of the authorities most widely recognized in human medicine.

■ Veterinarians employ rectal injections with the hind quarters of the dog elevated. Small dogs are usually kept in a standing position, and the pressure level is governed mostly by the height of the most convenient nail on which the container for the fluid is hung. In administering a lavage one must consider that there are other factors present in conjunction with the fluid. These are pressure, temperature, motion and osmosis, which may act to influence normal and disturbed physiologic processes in the gastrointestinal tract. Lavage should be employed under low pressure, a two-foot elevation of the container being enough so that the defecation reflex is not stimulated until sufficient fluid enters the bowel. The fact that a dog readily vomits a copious enema places him in a category different from man in respect to intestinal lavage. In man the direct effect of this therapeutic and diagnostic measure is con-

fined to the lower bowel, but in dogs it extends throughout the gastrointestinal tract, although only irregularly to the cecum.

According to Hall, injections of tap water cause an edema of the epithelial cells of the intestinal mucosa that sometimes require 48 to 72 hours to be overcome and during this period intestinal absorption is arrested. He also states that saline solutions do not have this effect. There are certain conditions where this arresting of absorption through the intestinal mucosa is highly desirable and consequently an enema of plain tap water justified. However, it implies an injury and suspended function ordinarily to be avoided, and certainly is contraindicated in lavage for diagnostic purposes. Except to arrest absorption, saline solutions should always be the basis for rectal injections.

In treating ulcerative colitis various substances have been used. Hurst advised the use of one to two grains of tannic acid to the ounce. Potassium permanganate (1 to 10,000) has also been used. Strong antiseptics must never be used as they will be absorbed. Bismuth subgallate (5%) suspension in olive oil is very useful; four to six ounces should be given and the dog forced to retain it if possible. Hypertonic salt solution, two drams of salt to the pint, tends to increase the flow of lymph from the ulcerated area and to stimulate granulation. The solution should be as nearly as possible at blood temperature and should be introduced slowly and without pressure so that spasm is not set up in the colon. To be effective the lavage should be done two or three times a day.

Coccidiosis has been treated with success by a lavage containing a tablesponful of a 5% mercurochrome solution to one quart of hypertonic salt solution. A quart, given at two-day intervals, over a course of four treatments, has proved effective.

■ The "massive" lavage is indicated for the removal of small foreign objects when their location is known to be above the pylorus. The intestinal tract should be first washed with an enema and the dog permitted to defecate. Copious amounts of a hypertonic salt solution are then given until the animal vomits the foreign material from its stomach. This method has obviated the necessity for surgical intervention in numerous cases. For the "massive" lavage a solution of three drams of sodium bicarbonate encourages emesis more promptly than salt water. Motility of the gastrointestinal tract being under control of the vegetative nervous system with the vagus the inhibitory nerve, the sympathetic the accelerator, some authors have attempted to explain the movements of the stomach and intestines upon a basis of a balance of these two sets of controls. Cannon, however, showed that the pylorus opened when the reaction was alkaline on the duodenal side. This may explain the reason of the more rapid action of the sodium bicarbonate lavage in causing vomiting.

However, on the general principle that the simplest therapeutic measure that offers promise of being effective should always be used in preferance to a more drastic one, emesis for the removal of foreign bodies from the stomach will be induced ordinarily by oral or hypodermic administration rather than resorting to lavage of the whole intestinal tract by rectal injection. If the simpler measures prove ineffective or if it appears that they will probably be ineffective, more drastic measures are of course indicated.

When the foreign object is known to be lodged below the pyloric cap it is dangerous to resort to massive lavage. Mills, working with clinical material, has shown that obstruction either organic, or caused by a foreign body at any point in the canal tends to cause stasis above it. A knowledge of this fact is important. All normal movement below the pylorus is downward and can be encouraged by a lavage of gentle action. To cite one example: A Scottish terrier, whose owner discovered a large handkerchief missing, was given an opaque enema and fluoroscopic examination disclosed an impacted mass in the intestines. Two quarts of flaxseed gruel was given as

a lavage and within 15 minutes an end of the lost handkerchief appeared at the anus. Gentle traction recovered the handkerchief in its entirety, as it had been swallowed without chewing. If any attempt to dislodge this object by massive lavage had been made, an impaction would have been probable.

■ The technique of use of the barium for the radiography of the colon is as follows: Mix one heaping teaspoonful of powdered gum tragacanth in one ounce of alcohol. If an electric beverage mixer is available, place one pint of very hot water in the container and while it is being agitated or stirred slowly, add the tragacanth and alcohol mixture. Let this mix well until a smooth, even paste is obtained which should be entirely free from lumps. Then fill a pint measure with powdered barium sulphate. Add this to one quart of hot water, then mix this barium suspension with the tragacanth paste. If any lumps or masses are present they must be removed by straining through cheesecloth; otherwise the tube used to give the solution will be plugged. The quantity of the enema may be increased to two quarts by adding sufficient warm water. An excellent suspension of the barium will then be obtained as it is important that an even distribution of barium through the entire mixture be fairly constant to produce the best diagnostic results. While this amount (two quarts) is excessive it is best to have that amount on hand. Administration can be stopped when the fluid appears to have reached the cecum, unless it is blocked by some obstruction of a foreign body or pathological condition. These enemas should always be given with the patient on its side and the flow of the opaque enema carefully watched by means of the fluoroscope.

■ Complete intestinal lavage is an important diagnostic procedure. Frick[3] discusses its value as follows:

"The average dog brought to a veterinary hospital needs an enema for the following important reasons. First: Many of them are decidedly constipated as judged by the history of their frequent vomiting, difficult defecation and symptoms of mild toxemia. The fecal findings show hard, concretive bowel contents. An enema to such a case brings immediate definite relief and is superior to the purgative of doubtful action. In large cities the percentage of cases of constipation is less than in towns and rural sections. Second: Where the patient is not constipated the information gained by the veterinarian from the enema is exceedingly valuable; oftentimes surprising information not obtainable in other ways is revealed. The feces from the enema should be examined for color. Is it black, red clay, white or other color? Is this color due to a drug administered? Bismuth subnitrate in large enough doses causes feces to appear coal black. Phenolphthalein makes them pink. Blood may be the reason for the red stool. Lack of bile is the common cause for a clay color. Large bone consumption tends to make the feces white. The consistency of fecal masses should be noted. Are they sticky, pasty, thin and liquid, or dry, granular and hard? The interpretation of the meaning of the consistency should check with the other findings. The odor should be given consideration. Is it putrid? Is the so-called 'distemper feces' odor recognizable? Tæniasis can be recognized readily by its characteristic odor. Check the chemical reaction for acidity or alkalinity, if advisable. Note if hair is present in the fecal deposit; or undigested peanuts, seeds, string or other foreign material. Are there tapeworm segments present? Note if there be a high gravel content. Valuable clues and much definite information will oftentimes be apparent to the naked eye."

Whitney's recent research[4] into the anthelmintic value of hydrogen peroxide has added greatly to the importance of complete intestinal lavage if the use of this agent should prove generally as effective in practice as it proved in Whitney's controlled experiments and has since proved in his hands and in the practice of others on field cases[5]. According to Whitney, hydrogen peroxide is effective in the removal of intestinal parasites of the dog only when administered rectally in sufficient volume to be vomited, and when so used eliminates nearly all the parasites; tapeworms, ascaris, hookworms, and even whipworms.

[3] Frick, E. J., 1934. The Enema and Fecal Examination in the Dog. *Vet. Med.* 29:3, pp. 130-31.

[4] Whitney, Leon F., 1939. Hydrogen Peroxide as a General Anthelmintic for Dogs. *Vet. Med.* 34:9, pp. 560-66.

[5] Magrane, H. J. and W. G., 1939. Hydrogen Peroxide as an Anthelmintic. *Vet. Med.* 34:10, p. 614.

A Survey of Wild-Animal-Disease Education In North American Veterinary Colleges

By JULIUS S. KATZ, Columbus, Ohio
Ohio State University

THERE is a branch of veterinary medicine knocking at the door of veterinary educational institutions to be admitted in its own right. The disease problems in the field of captive and free wild animals are being offered to the profession for investigation.

The demand for veterinarians trained in wildlife disease, comes from wildlife managers, wildlife researchers, fur farmers, zoo managers, and conservationists.

Wisconsin, Ohio, Michigan, New York, and California employ veterinarians whose work is primarily in wildlife conservation. Several men devote all their time to diseases of zoo animals, and sanitation in zoos, while others, as part of their practice, conduct periodic check-ups on the health of the menageries. The fur-farming industry is demanding veterinary service. A few of the larger ranches hire veterinarians to devote their energies to overcoming losses among their stock. The smaller farms are using the services of local practitioners.

Thirty-three colleges in the United States offer either partial or four-year training in wildlife management and conservation. Graduates in wildlife management are a group devoted to the study and manipulation of factors which influence wild animal populations. Disease is one of the factors. Eight of the thirty-three colleges containing these wildlife groups are located at schools that provide also veterinary training. Thus, in colleges containing eight of the ten veterinary institutions in the United States, there are groups of men who are being trained in wild-animal husbandry and are finding disease problems that demand solution.

Educators in live stock diseases have recognized the importance of including animal husbandry in the veterinary curriculum. There are, however, management techniques and practices, and also diseases peculiar to wild animals. A course including such wild-animal fundamentals added to veterinary training would enable the graduate to apply his medical training more intelligently to wildlife problems.

■ A survey was undertaken to learn how much wildlife disease training is being incorporated in the veterinary curriculum. Is the graduate prepared to utilize his medical training in problems presented by wild-animal husbandry groups?

A letter of inquiry was sent to 11 veterinary colleges in the United States and Canada. Two questions were asked:

1. What courses or parts of a course deal with any phase of wild-animal diseases, and are offered to:
   (a) graduate and undergraduate veterinary students?
   (b) graduate and undergraduate wildlife-management students?
2. Are you planning any wildlife-disease courses for the future, to be offered to (a) or (b) groups in question 1?

The following was received from the 11 veterinary colleges[1]:

Seven (Alabama, Colorado, Kansas, Michigan, New York, Ohio and Washington) make references to wild-animal diseases in several of the established courses of parasitology, pathology, and bacteriology. The extent of these references was not definitely indicated

Four (Alabama, New York, Ohio and Ontario) carry on limited diagnosis with wild animal specimens.

Three (New York, Ontario, and Pennsylvania) invited outside lecturers to deal with wild-animal diseases.

Two (Pennsylvania and Texas) offer a specific course in wild-animal diseases.

One (Iowa) offers a graduate course in wild-animal diseases, open to veterinary and advanced science students.

Two (Colorado and Ontario) offer short courses for students and laymen interested in fur-farming.

One (Ontario) conducts extension work for the benefit of the fur-farmers.

[1] The writer wishes to thank the deans and acting deans of the veterinary colleges of North America for their replies to his inquiries.

Two (Michigan and Ohio) plan a wildlife disease course in the near future.

■ Several expressions of interest accompanied the survey data. Some individuals felt that a course in wildlife disease is worthy, but that the veterinary curriculum is already overcrowded with fundamental work. As a solution to this, it was suggested by others that such a course would better fit the graduate student who wishes to specialize. This, however, would not prepare the general practitioner who may locate in a community that includes fur-farm practice, zoo, or conservation wildlife work.

Seven of the veterinary colleges reported inclusion of wildlife disease training in the regular courses of parasitology, pathology, and bacteriology. This correlation, as one individual stated, "gives better fundamental disease relationships than special classes." The references made in class depend upon the particular interest of the instructor in wild-animal diseases, and the information he may have at hand. The majority of references to wild-animal diseases in veterinary courses are made in order to describe more completely, or to parallel, a condition which exists in domestic animals. Material available for wildlife disease training is widely scattered in isolated articles and reprints. There are at present few libraries, except personal collections, that have sifted out wildlife-disease references. There is a fair quantity of material available for classroom work; this is being compiled at the present time.

In three replies it was stated that most diseases of wild animals are also found in domestic animals, implying, perhaps, that the veterinary curriculum is sufficient for wild-animal work. Yet, Pennsylvania and Texas are offering special courses in wild-animal diseases, and Ohio and Michigan are planning to do so in the near future.

■ What is the reason for these somewhat opposing thoughts? The group or individuals who feel that references to wildlife diseases in other courses are sufficient may be considering the similarities, while those who feel the need for a separate course may be noting the differences between the diseases of wild and domstic animals. For, although various wildlife diseases are found among live stock and domestic fowls, there are several maladies peculiar to wild animals. The environment of wild animals is different from that of domestic live stock, and this results in some differences in the epizootiology of wild-animal diseases. Under captive conditions the feral animal in its disease problems resembles more closely the domestic animal. Even under semi-domestic conditions, however, there are unlikenesses in feeding, breeding, and living requirements. The methods of handling (restraint), drug administration and drug dosage also may differ.

An elective course, that might be considered a survey of wild-animal diseases, could take account of these differences. The general practitioner, as well as the specialist, could be prepared fundamentally thereby to undertake wild disease problems, and so be able to apply his veterinary training with better understanding.

The veterinarian frequently depends upon the wild-animal husbandmen to collect and ship material for diagnostic work. Thus, a course offered by the veterinary colleges to wildlife groups may well favor close cooperation in wild-animal disease problems. This type of extension work has already proved successful at Colorado and Ontario schools which offer short courses for laymen and students interested in fur-farming. Iowa is another school to report such training for wild-animal husbandmen in a graduate course offered to students in advanced science.

■ The position which the veterinarian will hold in the study and control of wild-animal diseases will depend, in good measure, upon the preparedness of future veterinary graduates with respect to wild-animal diseases and wildlife, in addition to their present special training. The preparation for such veterinary training should be taken out of the field of individual interest, and placed in the organized curricula of veterinary medical institutions.

Diseases of Game Birds

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Disease Among Wild Birds

Previous studies have shown that practically all of the common diseases of domestic poultry may infect one or more species of game birds. To what extent outbreaks in the wild may account for widespread losses is not known, but in some types of environment and with certain combinations of factors disease may cause appalling waste. For long a cyclic or periodic disappearance of grouse, covering a period of about 10 years, has been observed. Since excessive rainfall during the nesting season, unusual numbers of predatory birds and mammals, or changes in the food supply do not satisfactorily account for this mortality, the presence of a contributory pathogen may be assumed, but as yet the evidence of this is unsatisfactory and incomplete.

Although wild bird populations are not comparable in density to those on game farms, the abundance of biting insects makes the spread of infections a simple matter. The fact that losses in the wild occur most extensively when populations are at their peak indicates that some contagion, unhygienic condition, or food factor may be involved. Sick or dead birds are especially difficult to locate in their native environment, and a carcass seldom remains long after death because of the action of scavengers and decay.

Disease on Game Farms

Disease conditions on game farms are very similar to those on the average poultry farm. To improve them proper sanitation and feeding must be observed because many of the losses on game farms are due to inappropriate feed and improperly arranged pens and houses. The most successful gamebird producers no longer try to maintain their stock in a natural environment but have developed a highly artificial, sanitary system of laying pens, incubators, brooders, and growing pens, as well as a series of composite feeds unlike those obtainable in nature. Even with these modern facilities diseases occur at times and are likely to be more destructive than among domestic poultry because of an apparent lack of resistance among wild birds in captivity.

J. E. Shillinger

Specific Infections

On game farms avian tuberculosis has been observed among pheasants, chukar partridges, wild turkeys, bobwhites, California valley quails, and various species of waterfowl. The practice of incubating and brooding game birds under domestic hens is responsible for many infections. Progress of the disease is relatively slow and a large proportion of a flock may be affected before a diagnosis is made. Among pheasants maintained for propagating purposes, tuberculosis may be carried over from year to year. Whether these birds are more susceptible than other species is not known, but the most extensive lesions observed by the authors were in pheasants. Although the liver is regularly the principal site of macroscopic lesions, all of the organs in the posterior part of the body cavity as well as the skeletal muscles, ribs, vertebræ, and sternum were affected.

Predominating symptoms are emaciation, weakness, pallor of the skin, and lameness. Avian skin does not respond satisfactorily to the intradermal test and, since most of the game birds have no comb or wattles where a more favorable type of skin exists,

the tuberculin test is of little value in diagnosing the disease.

Ulcerative enteritis, commonly known among game farmers as "quail disease," is probably the most difficult of the infections to cope with. It is not known to affect pheasants, but it is very destructive to quails and grouse; immature birds appear to be most susceptible. Young turkeys are reported to have an identical or very similar disease.

Various investigators have attributed this malady to several organisms. Bacteria-free filtrates have never reproduced the disease, but apparently pure cultures of *Corynebacterium perdicum* have produced a few cases in test birds. Whatever the causative organism may be, its maintenance in laboratory birds for more than a few successive transmissions is difficult. Feeding ground-up intestinal tissues or intestinal contents from affected birds, is the usual method of producing the disease in laboratory birds. Elsewhere the infection is generally spread through droppings or through contamination carried by attendants or flies.

The lesions most commonly observed in ulcerative enteritis are in the small intestine. These may show, in early stages of the disease, only as petechial hemorrhagic areas in the mucous membrane, but in extensive ulcerations may later develop in the intestine, often completely penetrating the wall and permitting the contents to flow into the body cavity. In well-developed cases the ulcers may be distributed throughout the entire length of the small intestine and the resulting inflammation causes extensive adhesions to adjacent tissue. The center of the older ulcers develop a yellowish caseous core. Some game breeders prescribe various medicinal preparations for the treatment of ulcerative enteritis, but most producers recommend prevention of the disease by thorough sanitation, including the control of flies by screening or trapping. As a remedial measure, keeping the birds in a warm place improves their chance of recovery.

Quail rhinitis, or catarrh, referred to by game breeders as "snuffles," is an acute, highly fatal, respiratory disease of young bobwhites. The cause of the condition is unknown although the manner of spread in certain pens suggests an infectious agent. Attempts to transmit the disease to healthy birds in good environment by use of nasal secretions have failed.

In the quail chick between one week and three weeks of age symptoms are frequent coughing and sneezing. The beak is held open much of the time and difficulty in breathing is evident. Autopsy reveals an excess of mucus in the nasal passages and larynx.

Losses from rhinitis in young quail usually begin during a hot period or at times of pronounced change in weather. The disease may be described as similar to a common cold in human beings.

Enterohepatitis, apparently identical with blackhead of turkeys, takes a more or less sporadic toll on quail farms. Occasional losses may occur at any time among birds of both sexes, but the most common incidence is among hens during the laying season. The disease is uncommon where brood stock is kept throughout the year on wire mesh, but losses may be considerable among valuable laying hens in ground-floored pens. So insidious is the malady that a hen may appear healthy and produce eggs in reasonable abundance up to a few days before death.

Lesions typical of the disease, as recognized in domestic turkeys, have been reported in bobwhites, wild turkeys, ruffed grouse, Hungarian partridges, sage grouse, and ptarmigans, although the circumscribed areas of degeneration frequently seen in the livers of turkeys are seldom found in the smaller game birds. So far as they may be applied, control measures are the same as those recommended on turkey farms. Exclusion of other birds, both domestic and wild, the use of wire-floored pens, and the exercise of thorough sanitation are the best known preventive measures. Investigators at the North Dakota Agricultural College found that blackhead in young turkeys on the rearing ground was prevented by dividing the flocks into small groups with four runs for each group, and a rota-

tion of occupancy practiced at monthly intervals. The houses and pens were portable and were moved to new ground each year in a five-year rotation. Since the cecum worms *(Heterakis gallinæ)* are known to carry the parasite causing blackhead, any measures which will remove them reduces losses.

Coccidiosis is of considerable importance on quail farms. Quail producers generally assume that the use of domestic chickens as brooders is objectionable because of the transmission of coccidia to the young quail. While losses from coccidiosis in quail are generally extensive, association with chickens cannot be held directly responsible. *Eimeria tenella, E. acervulina,* and *E. mitis,* found in domestic chickens, have been identified in quail, and *E. phasiani* and *E. dispersa* have been recognized in pheasants. *E. dispersa,* frequently occurring in the bobwhite, is not a parasite of poultry.

The modern method of raising game birds in pens with wire-mesh floors has aided in controlling the ravages of coccidiosis among the young. Keeping brood stock in such pens throughout the year is very effective in eliminating the disease.

Trichomoniasis is not infrequently found in quail. A foamy diarrhea of grayish-white or greenish excrement is the characteristic symptom and if it continues for a considerable period death occurs. Post-mortem examination usually reveals anemia and enlarged ceca. Flies are generally believed to carry the infection in nature and outbreaks on game farms have been checked by placing the birds in fly-proof pens.

■ Food poisoning presents a constant menace in game breeding, generally resulting from one of three causes: decomposition due to molds or fungi, decomposition resulting in the production of ptomaines, or the formation of toxins by bacteria. Feeds containing a high percentage of protein or dried milk, meat by-products, and fish meal are those most likely to be involved.

Of food poisonings due to bacterial growth, botulism is perhaps of greatest importance. Preformed botulinus toxin takes an annual toll of thousands of wild waterfowl. Three types of the botulinus organism, serologically distinct, are recognized in this country and are designated by the letters A, B, and C. Avian species are most susceptible to type C and sometimes suffer high percentages of mortality. Proper management of alkaline lakes on wildfowl refuges in the western States has resulted in considerable reduction in losses from botulism poisoning.

On the game farm, botulism in birds most commonly results from their having eaten fly larvæ or dermestid beetles that have fed on contaminated material. The botulinus bacteria are soil organisms that have a wide distribution. Any carcass may easily become contaminated from the soil and act as an excellent medium for the multiplication of bacteria and the production of toxin. Losses of birds on ranges not carefully inspected daily may pyramid because of constant addition to the number of decaying carcasses with increasing chances for getting the poison. Losses in young pheasants on range have been known to exceed 2,500 birds within a period of 10 days before the cause was found and removed.

Treatment with antitoxin in most instances is not practical because of the excessive cost of the serum. Flushing of individual birds with water is effective with wildfowl but seems of little benefit with the more susceptible gallinaceous species.

Scrupulous inspection of yards and runs, removal of carcasses, and prevention of food contamination offer the best solution to the problem of botulinus control.

Nutritional roup, from a deficiency of vitamin A in the food, may cause more or less extensive loss when an adequate ration is not provided. The characteristic lesions of the disease, grayish-white patches in the mucous membrane of the mouth, pharynx, and esophagus, are easily recognized. The kidneys and other visceral organs also may show somewhat similar grayish-white deposits on the serous surfaces.

Rickets and perosis, other nutritional diseases common on game farms, are similar to these conditions in domestic poultry.

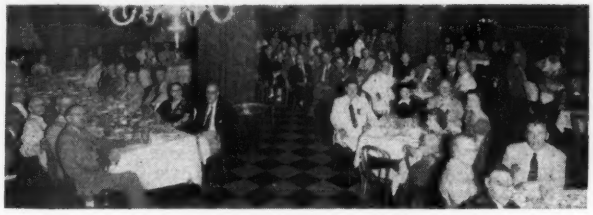
Cannibalism, or destructive pecking, is a common vice among game birds. The parts usually attacked are the head, the

vent, and the toes. The habit is probably due to close confinement or dietary deficiencies; changing the character of the feed sometimes has a beneficial effect. Some workers have had excellent results from the addition of an abundance of finely ground meat to the ration. Clipping the points of the beaks with sharp scissors and keeping the birds in semidarkness, except when feeding, also have served to reduce this trouble.

Conclusion

Most of the scientific studies of game bird diseases thus far conducted have been of birds kept on game farms. Many of the maladies observed on game farms, however, have also been recorded in the wild. What part these diseases may play in the decrease of game birds in their native environment has not been proved, but it is reasonable to assume that some of these pathological conditions are of importance in the periodic decreases in populations.

THE KANSAS CITY VETERINARY COLLEGE ALMUNI BANQUET



This banquet, held at the Memphis meeting of the A.V.M.A. last summer, was made notable by the presentation of a life-size oil painting of himself to Dr. A. T. Kinsley of Kansas City, a graduate of the school in 1904, assistant or professor of histology and pathology 1905-18, and president of the school 1913-18. Hugh E. Curry ('08), State Veterinarian of Missouri, was chairman of the committee that arranged for the painting; the presentation was made by D. M. Campbell ('07), Chicago. The banquet was attended by 132.

Infection of the Central Nervous System by Louping Ill Virus

Using the quantitative egg membrane technique, the authors[2] studied the infection of the central nervous system of the mouse by "louping ill" virus. They found that when the virus is inoculated intraperitoneally it usually enters the central nervous system *via* the olfactory mucosa and olfactory bulb, but not invariably. Trauma of the cerebral hemisphere of mice so inoculated allowed the virus to enter at the point of damage and accelerated the spread of infection. After intranasal inoculation, the virus entered the olfactory bulb and multiplied there, reaching a high concentration before the rest of the brain became infected. This suggested that there is a "physiological barrier" between the olfactory bulb and the rest of the brain. Once the infection passed beyond this barrier it spread rapidly down to the spinal cord and the virus was detectable in large amounts everywhere.—*T. S. Gregory.*

[2] Burnet, F. M., and Dora Lush, 1938. Infection of the central nervous system by louping ill virus. An investigation by the quantitative egg membrane technique. *Aust. Jnl. Exp. Biol. Med. Sci.* 16, pp. 233-240. Abst. in the *Vet. Bul.* 9:10, pp. 708-709.

Contagious Agalactia of Goats and Sheep

Two-thirds of the cases were of the chronic type with eye infections, arthritis, abscess formation in the lymph nodes and mammary glands and in the lungs and liver[5]. Induration of the udder was very frequent. The local breeds seem to be more resistant than imported breeds. The annual mortality from this disease during the years 1931-1936 was 6% in adult goats and 1% in adult sheep.

Every method of chemical or serological treatment tried was unsuccessful. The most effective method of dealing with the disease is the elimination of infected animals and a periodical examination of the herd. It is suggested that there may be some connection between contagious agalactia and worm infestation or that the environment may play some part in the incidence of the infection.—*Hans Graf.*

[5] Ratti, R., 1938. L'agalassia contagiosa delle capre e delle pecore in Val Bregaglia, Cantone dei Grigoni. *Schweiz. Arch. Tierheilk.* 80, pp. 317-320. Abst. in the *Vet. Bul.* 9:10, p. 709.

Dietary Diseases of Ranch-Raised Mink*

By J. A. NELSON, Vancouver, B. C.
Photos by E. E. Wegner

THE fur-farming industry in Canada has a most interesting and romantic history. In the early days raw furs were the only staple sought by Europeans in exchange for their products. The spread of the fur trade over the entire Dominion built the framework for our vast lumber, mining, and fishing industries. As these industries with their supportive population advanced with the fur trappers, so the available hunting grounds receded and dispersed until the pioneers in fur-farming saw the practicability of raising their pelters in captivity.

From a small start in Prince Edward Island over 40 years ago, fur farming has spread over all of Canada. In the last 20 years, and particularly the last five years, the growth of the industry has been phenomenal, until it represents an investment of over $20,000,000, and supplies 50% of the total raw fur production of Canada, totalling about $7,000,000 annually.

The silver fox is still the mainstay of the industry, but mink are rapidly becoming of parallel importance. There are more mink farms in the Province of British Columbia at the present time than there were fur farms of all kinds in the Dominion in 1919. During the last few depression years many ambitious persons, driven from other means of livelihood, have established mink farms, and unfortunately many lack sufficient knowledge to avoid even simple troubles, principally dietary.

There is not the wealth of experimental information from pioneer fox farms to guide the mink raisers, nor do they receive protection under the Feed Division of the Department of Agriculture as do the fox raisers. They are left wide open to exploitation by commercial feed companies and others, and it is the inexperienced beginners who are the most likely victims. It costs them more per animal for professional correction of a herd problem than the larger, well established rancher, and they grasp at any source of free advice.

There are indications that veterinarians are not fully recognizing the growing importance of the fur industry and equipping themselves to render efficient service when called. We never had, nor likely ever will have a better opportunity to gain and exchange knowledge on the various problems encountered in fur-bearers of all types than right now during this period of rapid expansion, when so many beginners are passing through their own trial and error stage. Their failures as well as their successes will have something to contribute to our accumulating knowledge. There has been a sad lack of exchange of clinical cases and problems of fur-bearing animals through the media of our journals, meetings and colleges. There are other sources of information that may not depend on fees for services which tend to eliminate the veterinarian, who is, or should be, far more capable of correcting these troubles. The more we can learn about these creatures the more quickly and economically can we render service and entrench ourselves as authorities, thus preserving this source of income for our profession.

At the present time nutritional diseases comprise the bulk of health problems of ranch-raised mink. True, there are other

*Presented at the Pacific North-West Veterinary Medical Association Convention, July 10, 11, 12, 1939.

causes of illness and death, but due to remarkable improvements in housing and management, and a deeper understanding of the unusual temperament of these creatures, these causes have been reduced to a minimum. In comparison with other classes of wild and domestic animals mink are apparently most resistant to infections. An occasional outbreak of a disease like hemorrhagic septicemia is about all that is encountered in British Columbia. Internal and external parasites are rarely seen; in fact if the proved methods of cage arrangement and handling are followed there is little excuse for parasitized mink. They are, however, very susceptible to fungoid infections which are sometimes contracted from the nesting material.

Mink are primarily carnivorous. Their food in the wild state is principally small game, birds' eggs, berries, leafy greens, small water clams, and fish. They are as much at home in the water as they are on land and have been observed to catch fish as large as themselves. Their chief starch intake is most likely from the partially digested contents of the stomachs and bowels of herbivorous game.

The old adage that an ounce of prevention is worth a pound of cure has no greater significance than when applied to mink. They are such notoriously unsatisfactory patients that the determination of methods to retain health are infinitely more important than the invention of methods to regain it. As with most wild animals their sense of self protection is so intensified that they resist the oncome of disease as long as they are physically able. As a result, by the time they are obviously sick the disease is far advanced and the chances of saving them greatly reduced. Furthermore, the value of an examination is minimized by fear which so masks the the symptoms that temperature, pulse, and respiratory observations are useless.

Enteritis

Enteritis is a common ailment of mink and is principally induced by the cereal portion of the ration. Their intestines are poorly adapted to the digestion of starch, particularly if uncooked. It seems surprising that mink may devour chicken and duck heads and whole small fish with impunity and yet develop hemorrhagic duodenitis from shredded wheat.

The causes of this condition are numerous, including excess of raw starch, coarse cereal, too fibrous vegetables, stale meat foods, sudden changes in feed, and overfeeding.

In cases where the majority of the herd develop diarrhea, gradually increasing in severity, the cause will usually be found in recent changes or additions to the ration. The evil of overfeeding lies in the mink's urge to hoard the excess to eat at a later time, when the food may no longer be wholesome. On occasions, usually on a holiday, an entire herd will be fed meat not known to be stale, often with disastrous results. Deaths may occur overnight, with many of the survivors emitting blood-stained and fetid vomitus and feces. Autopsy on typical cases of such poisoning show hemorrhagic areas on the stomach lining, less severe but more diffuse enteritis, hemorrhages into the pericardium and beneath the kidney capsules. Cultures from these areas have yielded *Staphylococcus aureus* and *Streptococcus viridans.*

Treatment is decided not so much by what is most suitable but by what is possible. Undue handling at this time may pre-

cipitate death so that drugs that will be taken voluntarily in food should be given, not forgetting that mink are highly sensitive or intolerant to many common drugs. Fortunately about the last thing a sick mink will lose is its appetite for raw beef or fish. I have obtained the best results by giving one tablespoonful of lean raw beef containing one-half teaspoonful of kaolin, or kaolin in metaphen, every three or four hours. Mink which have lost their appetite from acute enteritis have little chance of recovery, although they are usually so weak they won't fight too strenuously against medication. It is always worth treating them, however, and a suspension of kaolin in boiled skim milk may be given. If prostrated, a little brandy in milk may tide them over long enough to recover, although this is not likely.

Kidney and Bladder Stones

Kidney and bladder stones have long been a fox and mink breeder's headache, and only recently has the cause been satisfactorily determined. After numerous unsuccessful attempts at prevention, with the exception of a few misinterpreted successes, a real clue was finally provided through the effects of vitamin A deficient diets on experimental animals, particularly rats. In these animals bladder and kidney calculi were commonly found, but were significantly absent in controls.

In March, 1938, during the gestation period on one of British Columbia's larger minkeries, several pregnant females showed urinary incontinence, followed by abortion and death. Some cases showed mild lacrymation. Autopsy revealed mucopurulent metritis in some, but bladder and/or kidney stones in all. Cultures of the uterine contents lacked significance, the cases were in isolated rather than adjacent cages, and the breeding history indicated nothing common to any one or more males, so an infectious cause was discounted.

Forearmed with a knowledge of these conditions in experimental rats, and the occurrence of ophthalmia, the diet was considered for its vitamin A and mineral content. The cod-liver oil used was of high quality but the vitamin D was fortified out of proportion to the Vitamin A content. The dose was computed on a D unit basis with a resultant low supply of A. Bone meal constituted 7% of the total ration, 2% more than considered necessary for pregnant females.

Bone meal was reduced to 5% and Lederles' Vi Delta Liquid Concentrate was given to supply 1,000 units of vitamin per female in addition to that already given.

From that day on there has not been a case of kidney or bladder stone, or abortion, on this ranch. All the pelters (about 350) were autopsied last fall, and I failed to find one concretion.

Independent reports have substantiated avitaminosis A as the cause of stones and abortions in mink, with little reference to calcium and phosphorus intake. Experimental feeding trials would be necessary to determine whether mineral and vitamin unbalance act independently or together in causing concretions.

Kit Cannibalism

Considerable economical loss is far too frequently occasioned by the mothers destroying or eating their young. This act is observed in several different forms with different causes.

(1) Dietary deficiencies leading to the kits being still born or in a very weak state will often induce the mother to remove all traces of having kitted.

(2) Excessive bone meal causing too great skeletal development leading to dystocia will cause the mother to grasp the head of the unborn kit to hasten its removal. In such cases she usually does not devour them, they will be found outside the nest box with teeth marks on their head.

(3) Another type of this disturbance is not so easily accounted for. One day a female will appear with six or more glands and teats developed, indicating as many kits. A few days later the number may be reduced to two or three. I do not believe this is due to external disturbances, as in such cases the entire litter is devoured. It seems more likely due to a reduction in milk supply and the female reduces her

litter to the size she can accommodate. As I mentioned before these are highly temperamental creatures and obviously if the milk supply is low the kits are going to arouse her anger by nursing excessively.

(4) In the fourth group are those cases of cannibalism entirely due to external influences. The cause can usually be traced to some disturbance which the mother feels imperils her kits. Apparently she will carry the protective instinct to the extent of eating the kits to save them from harm.

Disturbances giving rise to this may come from too close contact of maternity cages. Further separation or a board partition between help eliminate it.

The worst case I ever saw was due to leaving the barren females beside the productive ones. In nine cases of cannibalism out of 159 litters there was a barren female in the adjoining cage, fretting and agitating because she was one of the "has nots."

After the removal of all barren females there were no recurrences.

Tail Biting and Tail Sucking

Tail biting or sucking is a common complaint from all over Canada and United States. Although a localized and seemingly simple condition, it is of considerable economic importance, causing unthriftiness, loss of pelt value and sometimes death. The mink bite, suck, or chew their tail anywhere from the tip up past the base. I have seen one case and heard of others where the mink in frenzy disemboweled themselves by tearing through the perineum.

Several logical theories have been advanced to explain the cause, all of which may be correct. Lack of calcium or bone meal has long been blamed and their increase in the ration has shown results.

Doctor Pyle suggests a deficiency of one or more of the vitamin B factors, with known results through adding a small amount of dried brewer's yeast.

However, the condition has been seen where there is apparently an adequate amount of these substances in the diet. Without contradicting either of these, I feel the origin of the condition lies in intestinal fermentation, acting either systematically on the skin, or locally through irritation of the anus. Removing, if possible, the cause of the fetid bowel condition and

administering intestinal astringents or antiseptics such as charcoal, metaphen or mercurochrome produces results, rather than the correction of a deficiency.

In dogs a similar condition follows irritation or congestion of the anal glands. It is possible that congestion of the musk glands in mink provide the stimulus to tail bite. However, this is the last part of a mink, dead or alive, that anyone cares to investigate.

Agalactia

A disease that has assumed considerable importance this year I have called agalactia for want of a better name. Identical cases have occurred from year to year but have heretofore lacked significance due their infrequency and apparent absence of cause.

It seems to be a more acute form of the condition inducing cannibalism through sublactation, although it occurs toward the end of lactation without cannibalism.

The predominating symptom is complete atrophy of all the mammary glands within two or three days, at about the 4th, 5th, and 6th week. The kits up to this time seem to be well nourished without taking more than usual outside food, indicating a sud-

den, rather than a gradual cessation of milk flow.

The kits usually are developed enough to carry on alone, but in all cases observed, numbering about 20 on various ranches, the mothers died after one or two days of inappetence and diarrhea.

Autopsy reveals the mammæ in an apparently dormant state. The carcass is

nearly devoid of fat, but not emaciated. The liver is mottled, which may be normal so soon after pregnancy, but not friable. The only other noticeable alteration is the flaccidity of the bowels, with no more than a suggestion of inflammation.

I believe there is a direct relation between this condition and the increasing price of liver. During the last few years spleens have been substituted to some extent for liver in the diet. As anemia is not a symptom, the cause is not associated with the anti-anemic property of liver or spleen, assuming the latter to possess it also.

In concluding his chapter on vitamin requirements for lactation Maynard, in his book on Animal Nutrition, states: "Recently several investigators have reported that there are lactation factors required by the rat which are different from any of the known vitamins. Most of the reports mention liver as a source of the factors in question."

Another possible cause for this condition is suggested in a report by Perla and Sandberg presented at the 1939 meeting of the American Society of Experimental Physiology, held in Toronto. I quote from the *Canadian Journal of Comparative Medicine*: "Experiments with animals have recently shown that an excess of Vitamin B_1 may develop disturbances in lactation and maternal instinct associated with cannibalism. This phenomenon was found to be due to a depletion of the manganese supply. On the other hand, an excess of manganese brings about the exhaustion of Vitamin B_1."

If vitamin therapy and increasing the liver intake have been of benefit I cannot say, because by the time treatment was initiated lactation was pretty well over. It now remains to hear reports from other localities or wait until next year to see if liberal liver feeding will prevent its occurrence.

Miscellaneous

There are a few food factors that merit mention, not because they have any detrimental or beneficial effect on health, but because they are thought to have an influence on the final pelt value.

1. Excessive amounts of fish cause the fur to lose color earlier in the season than normal, and may be a cause of fading of the pelt. Also, excessive fish will produce a heavy leather which is not desirable in the finished garment.

2. Excessive starch or carbohydrate feeding will cause singe, *i.e.*, curling of the tips of the guard hairs.

3. Excessive cod-liver oil or oil of high fatty acid content will cause rust, *i.e.*, reddish appearance in reflected light. This is really more noticeable in silver fox than in mink. For safety, wise ranchers do not feed cod-liver oil to pelters after the end of August.

4. Sodium chloride will cause the pelt to become dry and lose its lustre, as if the animal had been destroyed during fever.

5. Sunshine in a dry atmosphere will not affect the color, but sun refracted through fog or humid air is detrimental.

6. Ammoniacal fumes from urine and droppings have a very detrimental effect on the pelt color. This is most evident in solid-bottom cages and is one of the many reasons why the cage bottom should be of wire mesh and at least 18 inches above the ground.

Abstracts

Lip Scab of Sheep Transmitted to Man

The authors[1] give a detailed description of the disease in sheep, quote some published cases of human infection from sheep, and record a case of ecthyma in a shepherd whose sheep and lambs were nearly all affected. The shepherd developed small scabby lesions on his hands and neck which soon recovered under local treatment. The infection was transmitted experimentally to a healthy lamb by material collected from the shepherd, and from two of his affected lambs to two healthy lambs.—*J. E.*

Vitamin Requirements of the Pig

Pigs were fed on a synthetic basal diet which allowed optimum growth when 4% of yeast was added to it[4]. When the yeast was replaced by aneurin, riboflavin and nicotinic acid, growth ceased after three to five weeks, but on the addition of either the eluate or the filtrate fraction of Edgar and Macrae (1937), growth was continued at a third of its normal rate for four to six weeks, after which it again ceased. When both fractions were added, full growth proceeded for five weeks, after which there was a decreased rate of gain in weight, which corresponded to a relatively decreased appetite. Those pigs which were fed on the basal diet with aneurin, riboflavin and nicotinic acid plus the eluate fraction, developed a posterior paraplegia which was not cured when the filtrate fraction was added to the food. The pigs which received the same food supplemented by the filtrate instead of the eluate developed a microcytic anemia and became increasingly subject to epileptic fits; both of these conditions disappeared, however, when the eluate fraction was also included as a supplement.

In addition to aneurin, nicotinic acid and probably rivoflavin, the filtrate and eluate fractions of Edgar and Macrae are essential for the nutrition of the pig. It is probable that yeast contains still another essential which at present is unidentified.—*G. Williamson.*

[1] Oppermann, T., and G. Stumpke, 1937. Der Lippengrind (Ekthyma contagiosum) der Schafe und seine Uebertragbarkeit auf den Menschen. *Arch. Derm., Berl.* 176, pp. 337-346. Abst. in the *Vet. Bul.* 9:10, p. 708.

[4] Chick, Harriette, T. F. Macrae, A. J. P. Martin and C. J. Martin, 1938. The water-soluble B-vitamins other than aneurin (vitamin B_1), riboflavin and nicotinic acid required by the pig. *Biochem. Jnl.* 32, pp. 2207-2224. Abst. in the *Vet. Bul.* 9:10, p. 731.

Bang's Disease Vaccination

A report[3] is made on the use since 1933 of U. S. D. A. Bureau of Animal Industry *B. abortus* strain 19 in vaccinating all heifers born to an infected state dairy herd in California. (A preliminary report of the herd was published in 1938.)

As these heifers came into production, they were placed with the old, nonvaccinated cows, many of which were proved to be shedders of *B. abortus*. As the utility of the old cows has decreased, they have been gradually eliminated until now only 10 are left, one of which was still shedding *B. abortus* in 1938. No cows or heifers have been brought into the herd from the outside. The vaccinated animals now in milk production total 51, or 19% more than the total number in the old, infected herd in 1933. The vaccinated cows have produced 146 normal calves. There have been five abortions and five stillbirths among vaccinated cows, but inoculations and blood tests indicate that these were not caused by *B. abortus*. *B. abortus* of virulent type (not strain 19) was isolated from two of the vaccinated cows, neither of which aborted and one of which had a titer of over 1:50 when vaccinated at the age of 24 months. Continued recent tests have failed to demonstrate the organism in the milk or vagina of these two cows. There is no evidence that the vaccine organism was transmitted to any of the old, nonvaccinated cows in the herd by association with the vaccinated heifers. When vaccinations were begun, 17 unbred heifers between 12 and 32 months of age were given full vac-

[3] Haring, C. M., 1939. Results of vaccination with *Brucella abortus* strain 19 in an infected herd. *J. A. V. M. A.* 94:6, pp. 587-589. Abst. in *Exp. Sta. Rec.* 81:4, p. 573.

cine doses, and the vaccination titers of some of these animals did not recede to below 1:25 for about four years. No significant persistence of titers has occurred in the balance of the heifers vaccinated between four and 12 months of age.

Demonstration of Rabies Virus in Decomposed Brain Tissue

The identification of Negri bodies in a decomposed brain by microscopic examination is unreliable; a suitable animal inoculation method is therefore desirable.[5] Intracerebral injection of putrid brain tissue produces death of the test animal from intercurrent infection or brain abscesses. It is, therefore, necessary to remove the bacterial contaminants from the putrid material prior to inoculation.

Of several bactericidal agents tried, ether proved the most satisfactory because its extreme volatility permitted prompt removal from the material after the bactericidal action had taken effect. Experiments showed that ether in final concentration of 10% sterilized the contaminated brain tissue by exposure for two hours at 4°C., but had no effect on the virulence of the rabies virus in the tissue up to 18 hours' exposure.

The following technique for practical use was developed:

The central portion of the hippocampus major (cornu ammonis) is dissected out, and a small piece removed for microscopic examination and for glycerination. The remaining portion of the horn is emulsified in 10cc of nutrient broth and allowed to stand for one hour at room temperature. The supernatant liquid (after larger clumps settle) is pipetted into a stoppered 15cc centrifuge tube, and about 1cc of 10% ether added. The tube is then shaken thoroughly and allowed to stand in the refrigerator for two hours. After centrifugalization at slow speed the supernatant liquid below the ether layer is removed for inoculation into four test mice.

Nine grossly contaminated dog brains known to be positive have been examined by this method, and in each case a confirmatory diagnosis of rabies was made, in an average of 10 days. On one animal in which no satisfactory microscopic examination of the brain could be made because of gross decomposition, a positive report was made 11 days after injection of the ether-treated brain into mice.

[5] Sulkin, S. Edward, and Nathan Nagle, 1939. Demonstration of rabies virus in grossly decomposed animal brains. *Jnl. of Lab. & Clin. Med.* 25:1, pp. 94-98.

The Dog as a Natural Reservoir of Brazilian Typhus

Dogs in affected areas have shown positive Weil-Felix reactions; the susceptibility of dogs to the virus has been demonstrated, and they are hosts of the ticks responsible for infection. For these and for epidemiological reasons, the author[6] considers it very probable that the dog is a natural reservoir of the virus.

The Economic Importance of Dogs

The author[7] protests against the reluctance of the United States Department of Agriculture to expand canine research, on the grounds that legislative opposition would develop because the dog is not a "farm animal." He points out that the farm-dog population is at least 7,000,000, and that these dogs are valuable and necessary to farmers both as protection and in some cases (*e. g.* sheep-herding) as work animals. By elaborate computation of the total number of dogs in the United States, the amount of food they consume, and the agricultural products required in this food (all based on the author's estimates, since no statistical information on these points is available), he arrives at the conclusion that a total of at least 37,305,000 acres of land in the United States is being used to produce dog food. This is the equivalent of all the farm acreage in all the New England states, plus Pennsylvania, New Jersey, Delaware and Maryland. Assuming that this figure is approximately correct, the author reasons that the economic importance of the dog to the farmer is more than sufficient to justify expansion of canine research by the agricultural department of the government.

[6] Dias, E., 1937. O cao como provavel reservatorio do virus de febre maculosa brasileira. *Brasil med.* 51, pp. 1245-1247. Abst. in the *Vet. Bul.* 9:10, p. 710.

[7] Whitney, Leon F., 1939. An Open Letter to the Secretary of Agriculture. *Amer. Ken. Gaz.* 56:11, pp. 7-11, 190-193.